

/ALTRONEGEST INFLUENCES GROWTH, REPRODUCTIVE, AND CARCASS TRAITS IN  
MALE SWINE/

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## REVIEW OF LITERATURE

### General Introduction

In recent years, the skyrocketing costs of production have caused financial hardships for many pork producers. Producers and researchers are working to reduce these costs. Advancements have been achieved in energy conservation through use of more energy-efficient buildings, feed mixing systems, and waste disposal systems. Animal scientists continue to formulate more digestible diets that improve feed conversion while designing feeders to decrease feed wastage. Raising boars for meat production may be another method for increasing production efficiency.

It is well documented that boars will grow faster and more efficiently than barrows and gilts if their nutrient requirements are met. Boars have: 1) an improved feed to gain ratio, 2) fewer days to market, 3) a longer, leaner carcass, and 4) larger loineyes (Walstra and Kroeske, 1968). The latter two would be of great economic value if the Lean Guide marketing concept as proposed by the National Pork Producers Council were to be implemented.

Castrating male pigs at a young age to prevent the synthesis of the objectionable boar-odor steroids is a common practice. Castration compromises rate of gain and efficiency of feed utilization and increases carcass fat (Desmoulin and Bonneau, 1979). A substantial economic improvement in swine production might be achieved by the marketing of intact male pigs that were void of boar taint.



## Raising Intact Males

### Advantages for Intact Males

There are two goals for studying boar growth: 1) to enhance leanness and muscling of the animal, and 2) to increase efficiency of producing lean muscle. It is well established that boars produce carcasses with lower dressing percentages than castrated males because of less body fat. Boars were 12% leaner than barrows and loineyes were 12 to 25% larger for boars than for castrated males (Prescott and Lamming, 1967). Boars also converted feed to body weight to gain 10 to 20% more efficiently than barrows (Prescott and Lamming, 1964). These results are consistent with the findings of others (Blair and English, 1963; Baiburtcjan, 1963; Robertson et al., 1965). Knudson et al. (1983) found that boars had their greatest average daily gain (ADG) of 1.04 kg at a live weight of 87.3 kg, whereas barrows had the greatest ADG of .95 kg at 76.3 kg.

Walstra and Kroske (1968) concluded that boars have: 1) a more favorable feed conversion rate, 2) longer carcass, 3) less backfat thickness at the shoulder, back and loin, 4) a lower percentage of fat and a higher percentage of muscle, 5) higher percentage of ham, shoulder and other primal cuts, and 6) a lower dressing percentage than barrows.

### Methods of Raising and Feeding Boars

Many early researchers concluded that differences in weight gain between boars and barrows were not great (Charette, 1961; Luscombe, 1962; Teague et al., 1962; Tribble et al., 1965). Others (Winters et al., 1942; Bratzler et al., 1954) found intact males to grow slower than castrates. In contrast, Speer et al. (1957) and Prescott and Lamming (1964) suggested that intact males grow faster with improved feed efficiency only at higher levels of dietary protein than either barrows or gilts. This

conclusion was confirmed by Campbell and King (1982) and Tyler et al. (1983). This conclusion is logical since intact males develop a greater amount of muscle and less fat than castrates when evaluated at the same live weight (Wallace, 1944; Luscombe, 1962; Prescott and Lamming, 1964). Boars fed protein levels from 19.6% to 15.7%, and lysine from 1.16% to .76%, grew faster than barrows from 22.7 to 113.4 kg (Prescott and Lamming, 1967). Tyler et al. (1983) concluded that feeding more dietary protein to boars (approximately 3 percentage points higher than the current recommendation of 18% protein during the growing period) was beneficial and the optimal level was 20.9%. The 18% protein level was needed to maximize the percentage of lean cuts. Boars had loin eye areas 12 to 25% larger than those of castrates. This was consistent with results obtained by Mulvaney et al. (1983). On the higher protein diet, the boar's head (non-edible) was smaller and there was less fat in the leg. Campbell and King (1982) also suggested that boars needed at least a 17% protein diet with a .86% lysine level for optimal growth to 70 kg. This is consistent with the results obtained by Williams et al. (1984) who found that boars need an 18% protein diet containing .86% lysine through the growing phase and approximately a 16% protein diet containing .74% lysine through the finishing phase. When boars were fed diets that contained higher lysine content, they consumed 9% less feed (Williams et al., 1984).

Experimenting with production systems, Tonn et al. (1985) found that there was no difference in ADG between group-reared and individually reared boars from 12 to 27 wk of age. There was a tendency for individually reared boars to consume more feed and therefore have an increased feed efficiency. It also was observed that individually reared boars had a greater incidence of lameness.

## Why castrate?

In a review, Robertson (1966) stated that "the practice of castration of farm animals is closely linked with husbandry and breeding methods." He goes on to cite some advantages that are claimed historically: 1) prevention of indiscriminate mating of developing gilts with the ability to maintain controlled breeding systems, 2) elimination of the undesirable boar taint or odor that is liberated upon cooking, 3) suppression of secondary sexual characteristics of aggressiveness and libido, facilitating herd management during the finishing phase, and 4) marketing that is geared towards castrates.

Perhaps, the foremost reason for castrating males is the attitude of the meat packing industry toward boar carcasses. Personal communication with Ark City Packing, Inc. (Arkansas City, Kansas) revealed that market-weight boars are automatically devalued \$.22/kg (\$.10/lb). The reasons given were: 1) coarser hair which is more difficult to remove during processing, and 2) a reduced backfat thickness making trimming of the loin more difficult with a greater likelihood of scarring the loin during processing. It was interesting to note that nothing was mentioned of boar odor in these young market-weight intact males. Their carcasses were processed along with those of market barrows and gilts, and were sold as fresh pork.

## Disadvantages of Castration

The castration process has its drawbacks and some of these include: 1) a labor intensive process, 2) postoperative pain, hemorrhage, and the possibility of infection, 3) reduced possibilities for sire selection, and 4) setbacks in potential performance during recovery (Robertson, 1966). Desmoulin and Bonneau (1979) concluded that

castrates: 1) consume more feed, 2) are less feed efficient, and 3) have increased carcass dressing percentage. A higher dressing percentage is due to increased deposition of carcass fat along with lower relative weights of the head, hide, legs, and feet.

## Sexual Differentiation and Endocrinology

### Brain and Fetal Development

Ford (1982) concluded that male pigs could demonstrate female behavior in response to estrogen until increases in testosterone sensitized the brain and allowed male mating behavior to be expressed. Gorski (1979) stated that if reproductive development occurred in the absence of gonadal hormones, regardless of the genetic sex of the animal, functionality of the brain will be feminine in the mature animal.

Animals go through maturational stages during which they become sensitive to steroid hormones (estrogen and testosterone). Although the sex of a pig is determined at conception, differentiation of mating behavior in males is thought to be a sequential suppression of the potential to display female behavior while enhancing the potential to display male behavior (Beach, 1975).

The sensitive period for testosterone-dependent suppression of female behavior occurs postnatally in species with short gestation periods (i.e., mouse, rat and hamster). In species with long gestation periods, the period of sensitization occurred either prenatally (i.e., guinea pig and sheep) or perinatally (i.e., dog) (Goy and McEwen, 1980). From these studies, one would predict that the sensitive period for sexual differentiation in pigs occurs prenatally.

Ford et al. (1983) attempted to tie sexual behavior to prenatal testosterone

secretion; however, they were unsuccessful. It was concluded that there was a postnatal component as well as a prenatal component of sexual differentiation in the male pig. This was determined by the proportion of barrows treated with estrogen that showed decreasing intensity of the immobilization stance as age of castration increased. Pigs castrated at 4 mo of age or older showed very little response. These results suggested that reduction in female mating behavior was associated with the increase in testosterone secretion during pubertal development (Ford, 1982).

### Hormonal Cascade

The general hormonal status of the male has been reviewed (Turner and Bagnara, 1976; Amann and Schanbacher, 1983). The median eminence of the hypothalamus is the origin for most releasing hormones that are neurosecretions (Schanbacher, 1982b). Unlike sows, the hypothalamus of the boar is arrhythmic in the production of luteinizing hormone-releasing hormone (LHRH), which is released continuously (Kittok et al., 1984). Since a specific follicle-stimulating hormone-releasing hormone has not been isolated, a more appropriate term is gonadotropin-releasing hormone. Gonadotropin-releasing hormone is transported by the hypophyseal portal blood system to the adenohypophysis (Levine et al., 1982). This stimulates release of luteinizing hormone (LH), also known as interstitial-cell stimulating hormone (ICSH) in males, and follicle-stimulating hormone (FSH) (Kastin et al., 1969). Both hormones have the testes as their target organ (Schanbacher, 1982b).

Both FSH and LH play a role in the regulation of spermatogenesis in the boar (Schinckel et al., 1984b). Follicle-stimulating hormone stimulates development of the seminiferous tubules and biosynthesis of steroid hormone (i.e., estrogen) (Zirkin et al., 1980). It also concentrates in the Sertoli cells or nurse cells where it is essential for the final steps of spermatid maturation. Follicle-stimulating hormone also was shown

to stimulate restoration of spermatogenic activity in regressed germinal epithelium (Steinberger and Steinberger, 1974).

Luteinizing hormone localizes in the cells of Leydig, also known as the interstitial cells (Allrich et al., 1982). Male androgens, mainly testosterone, are synthesized in the Leydig cells within the parenchyma of the testes (Zirkin et al., 1980). Luteinizing hormone, FSH and the androgens are all needed for normal development and function of the testis (Schinckel et al., 1984b).

Testosterone and androstenedione are the main circulating androgens of testicular origin (Martin et al., 1984). Testosterone, through a feedback system to control the release of gonadotropins, stimulates development and maintenance of secondary sex organs and characteristics (Ford and Schanbacher, 1977). It also promotes protein anabolism by causing increased nitrogen retention in the form of tissue protein. Testosterone is bound to the serum proteins and thus normally does not filter through the kidney glomeruli (Turner and Bagnara, 1976). It is not stored in the body, but is utilized quickly or degraded in the liver. Degradation products of the steroidal hormones are not salvaged for reuse in the synthesis of new compound (Turner and Bagnara, 1976).

#### **Hormonal Changes at Puberty**

The intervals of peaks and declines of hormonal concentrations in serum historically have been reported to be correlated with weight gain or age. Researchers generally agree that there is an interdependence of weight as well as age on the timing of hormonal changes. During the early life of the boar, the predominant androgen is androstenedione and its biological potency is much lower than testosterone. The ratio of androstenedione to testosterone generally declines with age, with testosterone becoming the most significant androgen as the boar

reaches maturity (Martin et al., 1984).

In male pigs, increases in testosterone secretion are evident at three different stages of development: 1) d 35 of gestation, 2) the first 3 wk of life, and 3) after 4.5 mo of age (Colenbrander et al., 1978; Ford et al., 1980; Ford et al., 1983), and then sharply declined at 250 d of age (Allrich et al., 1983a). Bonneau and Desmoulin (1980) suggested that the final rise in testosterone corresponded to a body weight of 80 kg. Colenbrander et al. (1977) concluded that all the testosterone-dependent physiological and morphological changes that occurred in the male pig were related directly to concentrations of LH in serum, which show a comparable secretory pattern preceding that of testosterone. The major increase in LH concentration occurs between 70 and 126 d of age (Schinckel et al., 1984a), and precedes the period of most significant testicular development (Schinckel et al., 1984b).

Boars reach puberty when they are 110 to 125 d of age (Andresen, 1976a). This is the time of the final increase in testosterone production and the boar will experience puberty. Signs of puberty include increased incidence of ano-genital sniffing, side nosing, extension of the penis, and mounting without causing pregnancy (Barber et. al., 1980; Tonn et al., 1985). Most boars will reach sexual maturity around 180 d of age (Andresen, 1976a).

This increase in testosterone likely is due to a combination of the pubertal rise in LH and/or greater responsiveness of the testes to gonadotropins during this period (Allrich et. al., 1983b). It also has been determined that the metabolic clearance rate of testosterone steadily decreased with age (Christenson et al., 1983). By 250 d of age, after maximal growth of seminiferous tubules and sexual accessory glands has occurred, high concentrations of testosterone may no longer be required because concentrations of testosterone decline drastically in serum.

Control of LH secretion by negative feedback of gonadal steroids has been postulated in the boar (Kittok et al., 1984). Similar observations have been reported

for rams (Riggs and Malven, 1974; D'Occhio et al., 1982) and bulls (Schanbacher, 1981; Schanbacher et al., 1983a). It has been determined that testosterone inhibits the secretion of pituitary gonadotropins (McDonald, 1980). The existence of an operative negative feedback system controlling LH secretion in male pigs castrated through 160 d of age has been suggested (Allrich et al., 1983a). After 160 d of age, the negative feedback response was absent, indicating that decreased sensitivity of the negative feedback mechanism may have evolved. Presumably, these changes were the result of the removal of gonadal steroids. However, there is the feeling by some (Gerandai and Halasz, 1981) that there exists a neural pathway between the gonads and the central nervous system which influences the release of gonadotropins.

Turner and Bagnara (1976) summarized the current hypothesized controlling mechanisms of the negative feedback system. The classically accepted mode of regulation, the "long loop" feedback system, is controlled by hormones produced in peripheral glands, such as the gonads, adrenal cortex, and thyroid. New evidence has shown the existence of a second mechanism, the "short loop" or automatic feedback system. This mechanism is an internal regulatory system in which the specific adenohipophyseal hormones, such as LH and FSH, are the controlling signals. The location of receptor sites for the "long loop" system are located in the brain, median eminence, or adenohipophysis, and the receptor sites for the "short loop" system appear to be in the brain, but have not been demonstrated definitively in the anterior pituitary.

Varying sensitivity to the negative feedback mechanism may be an explanation for variable testicular size among individuals. Boars with heavier testes had higher pubertal concentrations of LH than boars with lighter testes (Schinckel et al., 1984a). These boars also tended to have higher concentrations of testosterone in serum during pubertal development and had greater sperm production (Schinckel et al., 1983). In these two studies, it was concluded that differences in testicular size



between boars with heavier and lighter testicular weight were independent of body weight.

Selection for increased rate of lean growth in swine reduced testicular weight and delayed sexual maturation (Schinckel et al., 1983). Although Andresen (1976a) did not measure testicular weight, higher levels of 5alpha-androstenone and testosterone were found in serum of boars selected for fatness and low rate of gain compared with boars selected for low backfat thickness and high rate of gain. Teague et al. (1964) found that fast-gaining, late-maturing boars slaughtered at lighter weights (82 to 95 kg) did not present a serious odor problem. This suggests a relationship between carcass and sexual maturation. One theory is that mammals must reach a particular ratio of body fat to lean muscle and total body weight before normal sexual maturation occurs (Frish, 1980).

Gray et al. (1971) suggested that physiological maturity rather than chronological age influences the rate of testosterone production in young boars. Martin et al. (1984) found boars had a near doubling of the testicular volume from 17 and 19 wk of age and was correlated ( $r=.48$ ) with a significant increase in serum testosterone.

### Boar Odor

#### Biosynthesis of Steroids Causing Boar Odor

Meat products from sexually mature boars frequently have an offensive odor upon cooking that has been described as "urine-like or perspiration-like" (Craig and Pearson, 1959). In a review of consumer responses concerning boar meat, Malmfors and Lundstrom (1983) concluded that most boar meat would be acceptable to consumers especially if boars were slaughtered at lower weights (80 to 100 kg).

Boar odor or taint is the common name given to a related group of  $C_{19}$ -16-ene steroid compounds and skatole (Bonneau, 1982). The main  $C_{19}$ -16-ene compounds include: 5 $\alpha$ -androst-16-en-3 $\beta$ -ol, 5 $\beta$ -androst-16-en-3 $\beta$ -ol, 5 $\alpha$ -androst-16-en-3 $\alpha$ -ol, 5 $\beta$ -androst-16-en-3 $\alpha$ -ol (Sink, 1967) and 5 $\alpha$ -androst-16-ene-3-one, which has the most intense odor (Desmoulin and Bonneau, 1982). The proposed pathway for androst-16-ene formation in the boar testis (Gower and Ahmad, 1967) and adrenal cortex (Gower, 1963) is: pregnenolone to progesterone to androst-4,16-dien-3-one to 3 $\beta$ - and 3 $\alpha$ -hydroxy-5 $\alpha$ -androst-16-enes. These then are converted to 5 $\alpha$ -androst-16-en-3-one (Berry et al., 1971; Thompson et al., 1972) and are taken up by the salivary glands (Patterson, 1968a) or stored in adipose tissue (Patterson, 1968b), especially the nonsaponifiable fraction (Craig et al. 1962), then released after a lowering of the plasma concentrations (Claus and Alsing, 1976). The remainder is probably catabolized in the liver (Claus, 1979). This would support the male odor hypothesis of Lerche (1936) that stated "if the testicles are functional, the causitive molecules will regularly enter the body and remain there even sometime after castration."

The characteristic boar odor has been found to be a sex pheromone (Sink, 1967) and has no anabolic effect (Saat et al., 1972). Before mating, the boar will salivate profusely liberating the odors of androstenone and other  $C_{19}$ -16-ene steroids (Melrose et al., 1971). The "mating stance" or "immobilization response" of females in estrus in response to pressure applied to the back (Altmann, 1941) has been shown to be induced largely by olfactory stimulation from these compounds (Signoret and du Mesnil de Buisson, 1961). Perry et al. (1980) found that by removing the submaxillary salivary glands to eliminate the white, frothy saliva, which normally contains the  $C_{19}$ -16-ene steroids, reduced the boars' ability to elicit a full immobilization stance from estrous gilts. It also reduced the boar's own libido.

The C<sub>19</sub>-16-ene steroids, androgens, and estrogen biosynthesized in the testis, are stimulated by LH (Carlstrom et al., 1975; Claus, 1979). These are secreted in closely parallel patterns (Claus and Hoffmann, 1980; McDonald, 1980). Even though testosterone and androstenone production are controlled possibly by the same mechanism, there is no interaction between the rates of biosynthesis (Cooke and Gower, 1978). Luteinizing hormone has been found to have stimulatory effects on the secretion of  $\Delta$ alpha-androstenone and testosterone (Andresen, 1975b; Carlstrom et al., 1975). This is the pathway by which sexual stimulation elicits increases in these two substances. Andresen (1975a) found that iv administration of hCG, which has LH-like activity, causes an abrupt rise in androstenone levels in plasma as well as in adipose tissue (Claus, 1976a). There was a rapid and delayed release of androsterone with the first peak occurring approximately 90 min after hCG and the second at 28 h postinjection. This pattern of release may be necessary to maintain sufficient reserves of sex pheromone for subsequent sexual activity (Claus, 1976a).

Vold (1970) demonstrated that skatole in boar fat had an intense fecal odor. Skatole is produced as the result of tryptophan degradation by microorganisms in the intestinal tract. With this in mind, it was suggested by Hansson et al. (1980) that skatole levels in fat are probably influenced more by environmental factors (i.e., diet, health) than physiological factors such as sex, castration, or genetics.

#### Developmental Chronology of Boar Odor

A marked increase in androstenone concentration in adipose tissue begins between 70 to 240 d (Andresen, 1976a) or a live weight of 80 to 107 kg (Bonneau and Desmoulin, 1980). These authors as well as Malmfors and Hansson (1974) agreed that androstenone concentrations in fat generally increased with age and live weight.

Andresen (1976a) indicated that there was a gradual increase in testicular secretion of androstenone which coincided with increasing concentrations of testosterone beginning at puberty, and that this increase continued until the time of sexual maturity.

In the adult boar, production of the C<sub>19-16</sub>-ene steroids is much higher than testosterone. While in the young boar, androstenone production does not prevail over testosterone. The ratio of androstenone to testosterone production seems to increase with age (Booth, 1975).

#### Sources of Variation in Tissue Content of Boar Odor

The influence of rearing conditions on adipose levels of androstenone was first described by Andresen (1975a) and later by Claus and Alsing (1976). They noticed a marked decrease in levels of boar odor when boars were moved from grouped pens to metabolism crates or individual pens. Claus and Alsing (1976) measured a transient increase in plasma and fat concentrations of androstenone in boars raised in confinement and moved to an open-front pen. Lightfoot (1979), Bonneau and Desmoulin (1980), and Narendran et al. (1982) observed a tendency for higher levels of androstenone in serum of boars reared in contact with gilts than for those raised in the absence of females. It also was noted that levels of androstenone in plasma and fat increased after excitement, such as fights with other animals (Liptrap and Raeside, 1978), or sexual stimulation (Andresen, 1976b; Claus and Alsing, 1976; Lundstrom et al., 1978), whether olfactory, auditory, or visual.

The idea that the presence of female pigs causes an increase in the concentration of testosterone is supported by the findings of Hemsworth et al. (1977). Stimuli from the presence of female pigs are necessary to maintain high levels of

libido in the boar. Walstra (1979) confirmed the stimulatory effect of the presence of female pigs by showing that boars reared with gilts had higher concentrations of androstenone than boars fattened separately. Observations by Andresen (1976a) showed that when three boars were penned together that one boar in each group would have a higher 5alpha-androstenone level compared to his penmates. They suggested a possible effect of social hierarchy on an individual's level of androstenone.

Willeke et al. (1980) successfully selected two different strains of boars; one having low and the other having high levels of androstenone in fat. In a similar experiment, Wafler et al. (1982) found that 51% of positive androstenone-selected boars were unmarketable compared with only 9.7% of the negatively selected boars. These experiments demonstrated the possibility for genetic selection for low concentrations of androstenone in tissue. Jonsson and Wismer-Pedersen (1974) estimated the heritability of sex odor in Danish Landrace boars to be  $.54 \pm .32$ . A heritability of this magnitude indicates the possibility that fairly rapid progress could be made through selective breeding.

Since there is a close interaction between the  $C_{19-16}$ -ene steroids and androgen production, this type of selection may affect productivity adversely. Some possibilities for selection based only on low content of fat androstenone may be: 1) late sexually maturing animals, which would be unfavorable for reproductive performance, 2) boars with decreased potential for androgen production and possibly reduced growth performance and carcass quality, and 3) animals with low potential for  $C_{19-16}$ -ene synthesis while maintaining normal androgen production. The last possibility would be most advantageous.

As suggested earlier, the chance of a carcass having a high concentration of boar odor may be influenced by genetics. The incidence of 5alpha-androstenone in

tissue samples was reported to be somewhat higher in the British Landrace than in the Large White breed (Rhodes, 1971). Pietrain were found to have an especially high odor content (Bonneau et al., 1979). Malmfors and Hansson (1974) found that 20% of the boar carcasses of Swedish Landrace or Yorkshire breed were tainted, of which only 1% was strongly scented. Lundstrom et al. (1980) suggested that neither boar-odor intensity, 5alpha-androstenone, or skatole varied with breed.

To date, researchers generally agree that the number of boars scored as having undesirable odor increased with weight, but weight alone did not influence totally the intensity of boar taint. Malmfors and Hansson (1974) concluded that slaughtering boars at 70 kg was no guarantee that taint would not be present, nor that all boars were tainted at 130 kg. There is probably an interaction between physiological development, age, and weight of the individual animal.

#### Detection of Boar Odor

A unique characteristic of boar odor is the variation and possible sex-related ability of humans to perceive the scent. Berry et al. (1971) noted that some panel members were able to detect the slightest hint of odor, whereas others could not detect odor at any intensity. This phenomenon may explain the wide variation in responses of consumer test panels. Griffiths and Patterson (1970) discovered a sex-related difference in the ability to detect 5alpha-androstenone. They found that 92% of women, whereas only 46% of men tested could detect this odor. Claus (1977) stated that a concentration of androstenone of 1 mg/kg adipose tissue is required for human detection. Carcasses with tissue levels of 5alpha-androst-16-en-3-one of 1 µg/g fat were rated as having a slight boar odor and would not be usable for fresh pork products (Thompson and Pearson, 1977). In highly sensitive individuals, the

threshold level may be as low as .4 mg/kg. These same conclusions were made by Desmoulin et al. (1982).

If boar meat is consumed, it is important to have a method for rapid detection of offensive boar odors at the time of slaughter. Walstra and Matemen (1982) discussed the available methods that included: 1) soldering iron method, 2) gas chromatographic method, 3) radioimmunoassay method (RIA), and 4) enzyme-linked immunosorbent assay method (ELISA). Odor detection may best be applied in slaughter house situations. The measuring technique must be relatively inexpensive and allows a large number of samples to be tested in a reasonably short time. The soldering iron method fulfills those two criteria. The disadvantage is the need for two inspectors in the slaughter line that are highly sensitive to the boar odor to reduce the risk of an odoriferous carcass being passed. Additional evaluation of suspected carcasses after chilling also is necessary. Under modern abbatoir conditions, gas chromatography and RIA are not practical. The major disadvantage is delay between sampling and obtaining results, as well as expense. In addition, there is the disposal of radioactive waste when using RIA.

#### Incidence of Boar Odor in the Sexes

About 65% of all boars and 1 to 5% of all females and castrated males were reported to have carcasses with evidence of boar taint (Williams et al., 1963). Andresen (1975b) measured 5alpha-androstenone concentrations in plasma of boars, gilts, and barrows and found mean levels of  $1.3 \pm 15.9$  ng/ml,  $2.3 \pm .8$  ng/ml, and  $1.1 \pm .3$  ng/ml, respectively. In a later study, Malmfors and Hansson (1974) showed that only 20% of 500 commercially slaughtered boars had the unpleasant taint when the carcasses were tested using a modified soldering gun technique (Lundstrom et al.,

1973).

In addition to the primary site of production of the  $C_{19-16}$ -ene steroids in the testes, Gower and Ahmad (1967) showed that biosynthesis of those compounds also can occur in the adrenal cortex. This may explain the presence of the offensive odor in barrows, gilts, and sows.

Sink (1967) postulated that a greater intensity of odor precursors occurs during certain periods of the estrous cycle in sows and gilts. This agrees with later work by Melrose et al. (1971) and Reed et al. (1974) and may explain the belief of farmers and small slaughter-plant operators that butchering female pigs in estrus should be discouraged (Berry et al., 1971). Another explanation for the presence of boar odor in female and castrated-male carcasses may be attributed to abnormal sex phenotypes, intersexes, or cryptorchids (Bishop, 1969). Thompson and Pearson (1977) found cryptorchids to have a mean 5alpha-androstenone level of 1.56  $\mu\text{g/g}$  of fat that was less than the average value of 2.09  $\mu\text{g/g}$  for intact male pigs (Andresen, 1975b).

### Prevention of Boar Odor

Surgical Castration. Castration is the routine method in swine production for prevention of boar odor. As stated previously, surgical castration causes reduced gains and feed efficiency in comparison with the intact male pig.

Alternative methods of castration have been examined. Baiburtcjan (1963) proposed a procedure that consisted of removal of the testicular parenchyma through a lateral stab incision through the tunics. Variable results have been obtained in pigs (Baiburtijan, 1963; Robertson et al., 1965) and cattle (Robertson and Laing, 1965) using this approach. This is probably due to the inconsistency of the amount of testicular parenchyma removed during the operation.



Rhodes and Patterson (1971), experimenting with different methods of castration, found that any technique of castration would eliminate boar odor, but also produced animals that grew significantly slower. Their treatment groups included: 1) testicular parenchyma removed, 2) testes removed while leaving the epididymis, 3) complete castration, and 4) intact males. This research confirmed the superior growth rate of intact males compared with barrows.

Andresen (1975b) determined that levels of 5alpha-androstenone in plasma of market-weight boars fell to approximately 2.5 ng/ml by 4 d postcastration. Cliplef et al. (1985) found that it took 12 wk for boar-odor steroids to decline to nondetectable levels in boars weighing 177 kg at 446 d of age. Claus (1976b) reported there was no influence of the concentration of C<sub>19</sub>-16-ene steroids on its rate of clearance at the time the boars were castrated at weights of 90 to 97 kg, 140 to 160 kg, and 240 to 250 kg. There was an effect of age and live weight because the time for clearance increased from 3 wk to 7 wk from the lighter to the heavier boars.

Delaying castration beyond the age at which it is commonly practiced (1 to 3 wk of age) has been suggested as a means of enhancing performance. Grosse (1963) and Prescott and Lamming (1964) indicated that pigs castrated after 4 to 6 wk of age had improved rates of gain and leanness. Bublik and Gerasimov (1977), experimenting with Russian Large White boars and appropriate timing of castration, found that castration at 90 d of age had a beneficial effect on ADG and feed efficiency. Delaying castration until 3 to 4.5 mo of age tended to improve growth and carcass characteristics (Domanski, 1973). Castration at older ages has been successfully used to eliminate boar odor (Bratzler et al., 1954; Andresen, 1975a). Even with the possibility of improving performance, late castration is likely to be more hazardous and stressful for the animal. Disadvantages include possible setbacks in growth immediately after surgery and the labor requirement for castrating large numbers of

heavier weight boars.

Late castration in other food-producing animals has shown to be of little value. Carrol et al. (1963) showed that late castration (6 and 12 mo of age) in cattle had little effect on lifetime performance, but did produce a slightly leaner carcass. For fat lamb production, castration has been shown to be unnecessary (Robertson, 1966).

Castration with testosterone replacement therapy has been reported by Schanbacher et al. (1983b). It was found that testosterone treatment generally reversed the effects of castration of boars weighing 45 kg. A consumer panel judging objectionable odor reported that samples from testosterone-treated barrows had either a slight or nondetectable odor. It was concluded that testosterone can be replaced in the castrated male pig at physiological dosages to maintain the anabolic effects on muscling and leanness similar to intact boars and that treated barrows do not have the characteristic boar odors.

Chemical Castration. Another treatment receiving attention is chemical castration. In theory, the mode of action is to inject a substance into the testis to cause slow destruction and fibrosis of the testicular mass. This technique is generally used for larger animals. Using this method, it is possible to maintain anabolic concentrations of testosterone longer before the biosynthetic pathway is disrupted permanently. Proponents claim advantages of nonsurgical, chemical castration that include reduction in pain and stress, and elimination of hemorrhage, hernia, infection, myiasis, and other surgical sequelae (Koger, 1978).

Intratesticular injection of calcium chloride ( $\text{CaCl}_2$ ) has had limited success in calves, pigs, dogs, cats, kid goats, and lambs. Calcium chloride is a very caustic necrotizing agent and if injected perivascularly will cause discrete local dry gangrene. If an excess amount is injected, or if leakage from the tunica albuginea

occurs, dry gangrene of the scrotum would occur followed by sloughing and uncomplicated healing (Koger, 1978).

Experimentation with varied concentrations and volumes of  $\text{CaCl}_2$  in a variety of solutions (i.e., water, 99% ethanol, and 70% isopropyl alcohol) has not yet produced the optimal solution or dosage. Senger et al. (1977) found the use of  $\text{CaCl}_2$  to be very difficult, especially in animals in which the testes are held close to the body cavity (i.e., pig, dog, and cat). If leakage occurred, moderate to severe external scrotal necrosis resulted. Results in cattle have been more favorable. Koger (1978) observed that bulls had a juvenile scrotum and a docile steer-like behavior 2 mo after chemical castration. Pain, swelling, and discomfort lasted approximately 24 h with no other detrimental effects.

A commercially available product called Chem-Cast® (Bio-Ceutic Laboratories, Inc. St. Joseph, Mo.) has been used in cattle. This product contains 88% 2-hydroxy-propanic acid as the active ingredient. Calves are castrated chemically at a weight of approximately 70 kg. If properly administered, the testes and tunics are destroyed and castration is complete by 60 d.

Researchers have examined various other compounds for chemical castration. Intratesticular injection of chemicals such as cadmium chloride (Benkov et al., 1975), 17% acetic acid (Lyatnikov, 1980), 17% citric acid (Lyatnikov, 1980), and sodium chloride (J.A. Froseth, personal communication) have shown no significant advantage.

Immunization Against Boar Odor. An area of study that is receiving great emphasis is the elimination of boar odor via an immunological approach. The theory of this approach is to conjugate a large non-specie specific protein such as bovine serum albumin or bovine thyroglobulin to the steroidal compound 5alpha-androstenone. This conjugation product, which acts as an immunogen, is injected into the pig and is

recognized to be alien in the body. In response, the immune system produces humoral antibodies against the foreign conjugated protein. When 5alpha-androstenone is released naturally into the blood stream, it is bound by the circulating antibodies and becomes inactivated biologically. Thus, 5alpha-androstenone is unable to accumulate in adipose stores, but still allows the boar to have intact testes.

Varied results have been obtained by researchers using the immunization process. Claus (1975) actively immunized boars against 5alpha-androstenone, but found little difference in tissue levels of boar odor between treatment groups. Other researchers (Williamson and Patterson, 1982; Shenoy et al., 1982) also have been unsuccessful in completely eliminating the boar odor in meat. In these studies, sufficient antibody responses were obtained and proper binding to circulating 5alpha-androstenone occurred. The inadequacy of the procedure may be the failure of the bound antibody-antigen complex to be removed from the blood even though tissue levels of 5alpha-androstenone were reduced. This is one explanation for unacceptable odor levels in the meat samples. These are contrary to results obtained by Patterson (1979). At slaughter weights of 90 kg, immunized boars had a 30 to 90% reduction in tissue levels of 5alpha-androstenone compared with controls.

It has been demonstrated that location of the protein on the 5alpha-androstenone molecule is important for effective antibody formation. Williamson et al. (1985) bound bovine serum albumin (BSA) to the 5alpha-androstenone at the third or 11th carbon position. Greater antibody titers were achieved in the boars that received 5alpha-androstenone-3-BSA. There also was decreased accumulation of boar odor in fat samples of these boars compared with boars that were immunized with 5alpha-androstenone-11-BSA. At weights of 90 to 95 kg, the fat concentrations of 5alpha-androstenone for the control boars, those immunized with 5alpha-androstenone-3-BSA, and the boars immunized with

5alpha-androstenone-11-BSA were  $1.77 \pm .20$   $\mu\text{g/g}$ ,  $1.10 \pm .18$   $\mu\text{g/g}$ , and  $1.99 \pm .38$   $\mu\text{g/g}$ , and at weights of 115 to 120 kg, 5alpha-androstenone levels were  $1.81 \pm .22$   $\mu\text{g/g}$ ,  $1.17 \pm .19$   $\mu\text{g/g}$ , and  $1.74 \pm .46$   $\mu\text{g/g}$ , respectively.

Thompson and Pearson (1977) demonstrated that there are other C<sub>19</sub>-16-ene steroids other than 5alpha-androstenone that are partially responsible for the undesirable sex odor of boars. This is in agreement with earlier work by Berry et al. (1971) and may explain the presence of detectable odor in meat products even with proper immunization. This also is substantiated by the moderate ( $r=.27$  to  $.75$ ) relationships between 5alpha-androstenone levels and sensory panel scores (Williams et al., 1963; Thompson, 1975; Andresen, 1975b; Malmfors et al., 1978).

In those studies, immunization against 5alpha-androstenone did not impair growth or carcass composition. Williamson and Patterson (1982) discovered that using an immunization technique did not elicit increased circulating levels of LH or testosterone. This may explain the reason for no alteration in growth of immunized boars. An apparent increase in the total amount of androstenone extracted from the serum of the immunized boars may reflect the recirculation of immunoglobulin-bound androstenone.

### Hormonal Manipulation of Intact Male Pigs

#### Autoimmunosterilization

Researchers have demonstrated recently the capability of producing an antibody response in animals immunized against endogenous gonadotropic hormones. The method of preparation and mechanism of action is similar to the method described earlier for immunization against 5alpha-androstenone. In females, cessation of the

estrous cycle occurs, but in males, a temporary state of castration results.

Immunization of ewes against androstenedione and estrone (Smith et al., 1981) increased the ovulation rate by 30% and a 27% increase in number of weaned lambs from ewes immunized against estrone was realized. The androstenedione treatment resulted in a higher proportion of conception failures because of an increase in anovulation, anestrus, and embryonic loss. This counteracted the effect of increased ovulation rate. These results substantiate earlier work (Cox et al., 1976; Scaramuzzi et al., 1977; Martin et al., 1979; Scaramuzzi et al., 1980) demonstrating the effectiveness of immunization against these female hormones.

Immunization of female swine against gonadotropin-releasing hormone (GnRH) also has been evaluated (Esbenshade and Britt, 1985). All gilts became acyclic with the last estrus occurring 10, 9, 4, or 2 wk later. Concentrations of LH in serum of gilts immunized against GnRH began to decline steadily during wk 4 and reached non-detectable levels by wk 11. Nondetectable levels were maintained through the end of the 18-wk trial.

Inhibition of LHRH by active immunization of male rabbits (Fraser and Gunn, 1973), rats (Fraser et al., 1974), monkeys (Hodges and Hearne, 1977) and sheep (Clarke et al., 1978) prevented the release of the pituitary gonadotropins. Autoimmunocastration results in decreased secretion of testosterone, atrophy of testes and other sexual accessory organs, and aspermatogenesis.

Schanbacher (1982a) found that rams immunized against testosterone or LHRH had reduced growth rates. There was no reduction in size of testes, sperm production, or testosterone in serum of those rams immunized against testosterone. Substantial amounts of antibody were formed to lower LH and testosterone in serum of LHRH-immunized rams. Release of both LH and testosterone was blocked in LHRH-immunized group when challenged by iv administration of 250 ng LHRH.

Immunization against LHRH severely inhibited testicular growth.

In bulls, the main effect observed after autoimmunocastration was a temporary castration, manifested by lower concentrations of testosterone in serum, testicular involution, aspermatogenesis, and docile behavior (Robertson et al., 1982). Bulls were immunized at 28 wk of age and boosters given 9 wk later. Growth and feed conversion for the immunized bulls were superior compared with steers. Immunized bulls also had larger loin eye areas and less fat than steers. This temporary castration effect lasted approximately 6 mo. It was determined that an antibody titer of 1:1,000 was needed to obtain a reasonable period of temporary castration.

Kuenstler et al. (1985) found that immunization of boars against LHRH at 12 wk of age resulted in undesirable carcass characteristics. Immunized boars had a greater backfat thickness and lower percentage muscle than barrows at 24 wk of age. However, performance traits (feed intake, average daily gain, and feed efficiency) and other carcass traits (length, loin eye area, and hot carcass weight) were similar between treatment groups.

Prolonged treatment with GnRH agonist depressed normal gonadal function in rats, dogs, humans, and cattle (Melson et al., 1983). Greger and Stricklin (1983) found that the GnRH agonist (D-Ala<sup>6</sup>,desGly<sup>10</sup>ProNH-GnRH ethylamide) inhibited testicular function. Mature boars were treated with daily injections of 100 µg for 30 d. At the end of the trial, testicular size had decreased, and concentrations of testosterone and androstenone in serum had declined to levels similar to castrated male pigs.

### Estrogens and Estrogen-like Compounds

Substances with estrogenic activity have little effect on the growing boar. The effect of steroidal hormones on growth and fattening of pigs was first reported by

Dinussion et al. (1951) and Woehling et al. (1951) using implants of stilbesterol and testosterone propionate, respectively. Stilbesterol had little effect on growth even though controls consumed between 5.2 and 13.7% more feed than treated boars. Overshadowing the experiment were the detrimental effects of the stilbesterol implant. Prolapse of the uterus of the females and renewed sexual drive of the barrows were common occurrences. Stilbesterol or testosterone propionate had no effect on gain when treatments were imposed at an average weight of 20 kg. It should be noted that treatment groups contained only gilts and barrows that were castrated at a young age. Treatments greatly enhanced the development of the reproductive organs of both sexes.

Experimentation with diethylstilbesterol (DES) has produced varied results. Implantation of boars with DES reduced boar odor while maintaining the advantageous growth characteristics of the boar (Plimpton, 1965; Echternkamp et al., 1969; Newell et al., 1973). Christian and Turk (1957) observed that feeding 10 or 50 mg DES/d from a weight of 62 to 132 kg decreased boar odor, especially at the higher feeding level. Diethylstilbesterol (96 mg) improved rate of gain while delaying the development of sex odor for boars at a live weight of 70 kg (Teague et al., 1964) and altered the deposition of intramuscular fat, but had no effect on the area of the longissimus muscle (loineye). In contrast, Ockerman et al. (1981) demonstrated that 96 mg DES implanted in 70-kg boars produced an intermediate-type carcass with the DES-implanted boars scoring between the control boars and barrows in the areas of muscling, leanness, flavor, and presence of sex odor. A consumer panel found no difference in boar odor levels between the three treatment groups when slaughtered at weights of 82 to 95 kg, or 103 to 134 kg. It should be pointed out that DES implanting in swine or any other food-producing animal is not permitted by the Food and Drug Administration in the U.S.



The effect of estradiol-17 $\beta$  implantation in the growing boar was studied by Timmis et al. (1984). It was concluded that a 24-mg implant had no effect on ADG, feed to gain ratio, backfat thickness, loin eye area, or testicular weight when boars were slaughtered at 165 d of age (approximately 110 kg). Implantation reduced the intensity of boar odor even though the controls were judged as having a slight odor.

Use of a combination injection of female gonadal hormones was used successfully to eliminate boar odor. Zivkovic et al. (1982) reported that 90 to 95-kg (6 mo of age) boars were devoid of sexual odors when they were injected with estradiol and progesterone within 24 h after birth. Treated boars had a better feed to gain ratio and less backfat compared with control boars. Treated boars also exhibited a quieter more docile attitude than the uncastrated controls.

Zeranol is a derivative of zearalenone, a natural metabolite of the mold Gibberella zeae. Zeranol is a potent, nonsteroidal anabolic agent with little estrogenic activity (Tindall, 1983) and has been shown to improve weight gains and feed efficiency in steers (Ralston, 1978; O'Lamhna and Roche, 1984). Experiments using zeranol have been conducted using intact male food-producing animals. Greathouse et al. (1983) reported that zeranol-implanted bulls had improved ADG and feed efficiency. Calkins and Clanton (1984) found that implanted bulls tended to have increased carcass fatness. Meat products from bulls implanted with zeranol were more desirable than meat from nonimplanted bulls (Lamm et al., 1980). Implanted bulls had decreased weight and length of the penis and decreased weight, volume, and density of the testes. Decreased behavioral problems (i.e., butting, riding, and rubbing on equipment) were observed after implanting bulls every 90 d starting at or before 3 mo of age (McKenzie, 1983).

In ram lambs, zeranol has been shown to suppress testicular growth, but had no significant effect on ADG (Riesen et al., 1977). When implanted at 44 and 89 d of

age, gonadotropin synthesis was inhibited as shown by reduced concentrations of LH and FSH in serum.

Experimentation with zeranol implantation has only marginal effects in the growing boar. Denzer et al. (1984a) indicated that implanted boars were heavier at slaughter and had larger testicles than control boars. However, in another experiment (Denzer et al., 1984b), zeranol had no effect on weight gain or testicular size. Both studies showed that implantation had no effect on boar odor.

### Progestogens

Hormonal manipulation of males using estrogen and progesterone, reduces concentration of LHRH in serum with subsequent reduction in serum LH, and testosterone production by the interstitial cells of the testes (Turner and Bagnara, 1976).

Goodman and Karsch (1980) reported that progesterone decreases concentrations of LH in serum by acting at the hypothalamic level. The literature contains many examples of progesterone as an antiandrogenic substance. Progesterone has been shown to reduce male sexual behavior in ring doves (Erickson et al., 1967), pigeons (Murton et al., 1969), guinea pigs (Diamond, 1966), mice (Erpino, 1973), and gerbils (Griffo and Lee, 1973).

Wheaton and Mullett (1982) demonstrated that implanting ewes with 350 mg progesterone decreased concentrations of LH in plasma. Luteinizing hormone response to LHRH administration was reduced in the progesterone-treated ewes to 27% of that for control ewes.

The effects of progesterone implantation on ram lambs is cited by Echternkamp and Lunstra (1984). Rams were implanted at 2, 4, and 8-wk of age. At 12 wk of age,

implants were removed. Progesterone-implanted rams had lower LH concentrations than controls that lasted for 1 wk posttreatment. Testosterone also were lower for the progesterone-treated rams.

Reports by Busch et al. (1979) concluded that chloramadinone acetate (CAP, a progestogen), fed at a level of 30 mg/d for 70 d, produced a desexualizing effect in boars. Boars were group-reared throughout the experiment and at approximately 75 kg, treatment was imposed. It was found that CAP decreased boar odor, testosterone, testicular mass, and spermatogenesis. As time of treatment progressed, it was observed that the CAP-treated boars had a slow decline in sexual activity as noted by reduced incidence of fighting and a more docile behavior. At 114 kg (289 d of age), CAP-treated boars were slaughtered (80 d after the end of the treatment period). At this time, 90% of the control boars had sufficient boar odor to render the carcasses unusable. Of the CAP-treated boars, 30 of 40 carcasses had no evidence of odor, whereas seven were judged to have a slight odor, but still usable. Thus, 93% of the CAP-treated boars were considered to have usable carcasses by consumer standards.

Altrenogest, which is another synthetic progestogen, is being investigated currently for use in female pigs for estrous synchronization (Webel, 1978; Martinat-Botte et al., 1984) and may be available to swine producers in the near future. Altrenogest suppressed LH surges (Redmer and Day, 1981) and ovarian follicular development (Guthrie and Bolt, 1982), while estrus was synchronized after withdrawal of altrenogest from gilts and sows (Webel, 1978; Day, 1984). The current recommendations for estrous synchronization is to feed altrenogest for 14 to 18 d (Stevenson and Davis, 1982) at a daily dosage of 15 to 20 mg per head (Webel and Day, 1982). Therefore, altrenogest may have the potential to reduce concentrations of LH in serum of growing boars and possibly reduce testicular synthesis of

testosterone and boar-odor steroids. If this can be achieved, then altrenogest could produce a castration-like effect in boars similar to the effect of CAP (Busch et al., 1979). The influence of altrenogest or CAP on growth and carcass characteristics is unknown.

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BODY GROWTH AND TESTICULAR CHARACTERISTICS OF BOARS FED A  
SYNTHETIC PROGESTOGEN, ALTRENOGEST

ABSTRACT

A synthetic progestogen (altrenogest) was fed to 24 Yorkshire x Duroc boars to determine effects on body growth, testosterone in serum, and testicular characteristics. Boars from six litters (blocks) were allotted randomly to one of four treatment groups at 12 wk of age. Treatment groups were: 1) controls, 2) altrenogest fed for 3 wk, 3) altrenogest fed for 6 wk, and 4) altrenogest fed for 9 wk. Treatment began at 15 wk of age feeding a daily dose of 20 mg/boar. Although there were no differences among treatment groups for gain and feed intake, boars fed altrenogest for 6 and 9 wk tended to consume more feed and were less ( $P < .05$ ) efficient than boars fed altrenogest for 3 wk or controls. Boars fed altrenogest for 3, 6 and 9 wk had lower ( $P < .05$ ) peripheral concentrations of testosterone in serum than controls from 15 to 24 wk of age. However, testosterone increased after altrenogest withdrawal in the 3 and 6-wk treatment groups but did not reach control concentrations by 24 wk. Boars fed altrenogest for 9 wk maintained testosterone below 1 ng/ml of serum during the treatment period. Despite lower concentrations of testosterone in serum of altrenogest-treated boars, backfat thickness was similar to controls, perhaps suggesting a slight anabolic effect of altrenogest on nutrient partitioning. Testicular weights and volumes following castration at 24 wk decreased ( $P < .001$ ) linearly with increased duration of altrenogest feeding. Concentrations of testosterone and estradiol in testicular venous serum at castration were lower ( $P < .01$ ) in altrenogest-treated boars than in controls. These data demonstrate that feeding altrenogest inhibits both testicular growth and steroidogenesis of boars without altering body growth or backfat thickness.

(Key Words: Boars, Progestogen, Altrenogest, Body Growth, Estradiol, Testosterone.)

## INTRODUCTION

It is recognized generally that feeding intact compared with castrated males enhances muscling and leanness of the carcass, and increases efficiency of producing lean muscle (Prescott and Lamming, 1964; Desmoulin and Bonneau, 1979). However, feeding boars for meat production has been limited in most areas of the world because of: 1) possible indiscriminate mating of developing gilts, 2) objectionable odors or boar "taint" in the fat and meat, 3) male aggressiveness during the finishing phase, and 4) traditional acceptance of finished barrows by consumers and meat-packers (Robertson, 1966). Several of these factors are related primarily to an increase in testosterone concentration that occurs at about 105 to 120 d of age or just before puberty (Colenbrander et al., 1978; Ford, 1982; Allrich et al., 1982). Therefore, if testosterone secretion could be suppressed for approximately 1 to 2 mo, boars possibly could reach market weight (100 kg) before onset of the undesirable male characteristics described above.

Progesterone reduced testosterone-dependent male sexual behavior in ring doves (Erickson et al., 1967), pigeons (Murton et al., 1969), guinea pigs (Diamond, 1966), mice (Erpino, 1973), and gerbils (Griffo and Lee, 1973). Busch et al. (1979) found that chloramadinone acetate (CAP) fed for 70 d (30 mg/d), produced a desexualizing effect on boars. The majority of those boars had a reduction of 5 $\alpha$ -androstene (major steroid responsible for boar "taint") in meat and fat, and were classified acceptable for market. Altrenogest, another synthetic progestogen, is being investigated currently for use in pigs for estrous synchronization and may be available to swine producers in the future. Altrenogest suppressed effectively LH surges (Redmer and Day, 1981), and ovarian follicular development (Guthrie and Bolt, 1982), while estrus was synchronized after withdrawal of altrenogest from gilts and sows (Webel, 1978; Day, 1984). Therefore, altrenogest may have the potential to produce castration-like effects in boars by reducing testicular secretion of

testosterone through suppression of LH release without altering advantages of boars for lean muscle growth. The objective of our study was to determine the effects of feeding altrenogest on body growth, testosterone in serum, and testicular characteristics of boars.

## MATERIALS AND METHODS

### Experimental Design

Crossbred boars (Yorkshire x Duroc) born in March, 1984 were weaned at about 3 wk of age into an environmentally controlled nursery. A pelleted complete diet (18% protein; 1.25% lysine) was provided ad libitum for 2 wk. For the remainder of the study, a medicated, 18% protein (.95% lysine) milo-soybean diet fortified with vitamins and minerals that met or exceeded all NRC (1979) recommendations was fed. At approximately 12 wk of age, boars were moved into a modified open-front facility with solid concrete floors. Intact males of similar weights ( $n=24$ ) were selected from six litters and were blocked by litter with one boar from each litter assigned randomly to one of four treatment groups at 12 wk of age. Treatment groups included: 1) controls, 2) altrenogest for 3 wk (15 through 17 wk of age), 3) altrenogest for 6 wk (15 through 21 wk), and 4) altrenogest for 9 wk (15 through 24 wk). The dose of altrenogest fed was  $20 \text{ mg} \cdot \text{boar}^{-1} \cdot \text{d}^{-1}$ . The 20-mg daily dose was chosen because a similar dose was adequate to inhibit estrus in postpubertal gilts (Kraeling et al., 1981). Boars were separated into arbitrary heavy- and light-weight groups and assigned randomly within treatment to pens ( $4.6 \times 1.2 \text{ m}$ ) with three boars/pen and two pens/treatment for the duration of the study.

Boars were provided free access to feed during the treatment period except daily from 0800 to 1600 h. At approximately 1530 h, pens were cleaned and 1.35 kg feed containing 60 mg altrenogest in 15 ml soybean oil or the control diet containing only 15 ml soybean oil was spread on pen floors away from pen sprinklers in an area of  $2.5 \text{ m}^2$  to provide equal access to feed by all three boars. Any feed remaining on

the floor (<5%) after 30 min was added to feeder bowls and ad libitum feed was made accessible to boars. After termination of the 6-wk treatment (22 wk of age), altrenogest was mixed into the feed for ad libitum intake from 22 through 24 wk in the 9-wk treatment group. Altrenogest was provided at 20 mg·boar<sup>-1</sup>·d<sup>-1</sup> based on the estimated daily feed consumption during the previous 2-wk period. Sprinklers were activated when ambient temperature exceeded 29 C.

#### Measurements

Feed consumption of boars per pen and individual body weight were assessed biweekly to determine average daily feed intake, daily gain, and feed conversion until 24 wk of age. Blood was collected via puncture of the anterior vena cava at biweekly intervals between 0800 and 1030 h to monitor testosterone concentrations in serum. Backfat was determined ultrasonically (Ultrasonic Scanaprobe, Ithaco, Ithaca, NY) at the first rib, last rib, and last lumbar vertebra, prior to castration at 24 wk of age. Boars received 1 to 1.5 ml (im) of a commercial tranquilizer (.4 mg fentanyl and 20 mg droperidol/ml) 20 min before being anesthetized with 1 to 1.5 g sodium thiamylal. Testes were removed and blood collected from a spermatic cord vein to quantitate concentrations of testosterone and estradiol-17 $\beta$ . Testicular weight and volume (by water displacement) were determined.

Testosterone and estradiol in serum were measured by radioimmunoassays validated in our laboratory. Rabbit anti-testosterone (#250) obtained from Dr. G. D. Niswender (Colorado State University) was utilized at a titer of 1:20,000 to achieve about 45% binding of the labelled testosterone (1, 2, 6, 7-<sup>3</sup>H-testosterone; New England Nuclear, Boston, MA). Specificity of this antiserum for testosterone was reported by Gay and Kerlan (1978). Serum (100  $\mu$ l) was extracted with ethyl acetate (79% recovery). When 100, 200, or 400 pg testosterone (n=4 each) were added to boar serum, 111, 222, or 424 pg was recovered (r=.98), respectively. Serum curves paralleled standard testosterone curves (Sigma, St. Louis, MO, T-1500 in 95%

ethanol). Variable volumes (n=4 each) of boar serum (50, 100 and 150  $\mu$ l) assayed 5.2, 4.4, and 4 ng/ml, respectively. Assay sensitivity was 17 pg/tube and coefficient of variation of two assays was 11.8% (within) and 11% (between).

Serum estradiol-17 $\beta$  was quantitated in a single assay using antiserum (Estradiol-6, #3) donated by Dr. N. R. Mason (Eli Lilly and Company, Indianapolis, IN). Antiserum specificity was tested against estradiol-17 $\alpha$ , estrone, estriol, testosterone, and androstenedione at 50% binding inhibition of the labelled estradiol (2, 4, 6, 7- $^3$ H-estradiol-17 $\beta$ ; Amersham, Chicago, IL) and cross-reactivity was nil (<.001). Recovery of tritiated-estradiol after two extractions of 1 ml serum with 10 volumes of ethyl acetate was 75%. Addition of 25, 50, or 100 pg estradiol-17 $\beta$  to 1 ml boar serum yielded 28, 49, and 97 pg (r=.98). Parallelism existed between boar serum (.3, .5, .75, 1, and 1.5 ml) and standard estradiol curves (Sigma, E-8875 in 95% ethanol). Assay sensitivity was 5 pg/tube and the intra-assay coefficient of variation was 9.8%.

### Statistical Analyses

Data were analyzed as a completely randomized-block experiment including treatment, litter, pen, and all two-way interactions using Procedure GLM (SAS, 1979). Pen was used as the experimental unit for growth and feed intake characteristics. Biweekly testosterone concentrations were analyzed using a pseudo-split plot analysis for repeated measurements (Gill, 1978). Treatment effects for biweekly testosterone concentrations were tested by the boar-within-treatment mean square. Preplanned orthogonal contrasts and orthogonal polynomials (linear) were used to characterize and separate treatment means where applicable.

### RESULTS

Boars averaged 44 kg at the beginning of the study (15 wk of age) and 92.7 kg when castrated at 24 wk of age (table 1). No difference ( $P<.05$ ) in daily gain was observed during any of the 2-wk periods or during the entire treatment period.

Although average daily feed intake was similar among groups, boars fed altrenogest for 6 and 9 wk tended to consume more feed and were less ( $P < .05$ ) efficient than boars fed altrenogest for 3 wk or controls. Estimated daily feed intake varied from  $2.3 \pm .2$  to  $2.7 \pm .3$  kg when measured at biweekly intervals during the treatment period. In addition, treatment had no effect on average backfat thickness.

Testicular traits were affected markedly by treatment (table 1). All treatments were different ( $P < .05$ ) from controls for testicular weight and volume. Testicular weight and volume declined ( $P < .001$ ) linearly as duration of altrenogest feeding increased. For every week increase in altrenogest feeding, testicular weight declined ( $P < .001$ )  $31.7$  g ( $B = -31.7 \pm 7$ ) and testicular volume declined ( $P < .001$ )  $29.7$  cm<sup>3</sup> ( $B = -29.7 \pm 6$ ). As expected, testicular weight (as a percentage of body weight) declined ( $P < .05$ ) in a similar manner.

Testosterone concentrations in serum collected on alternate weeks from 12 to 24 wk of age are illustrated (figure 1). Testosterone concentrations for altrenogest boars were lower ( $P < .05$ ) 1 wk after initiation of treatment (16 wk of age) than controls. Results indicate that boars fed altrenogest for 3 wk had a delay of about 4 wk in the rise of serum testosterone concentration compared with the earlier rise in controls. Boars fed altrenogest for 6 wk had a similar delay (6 wk) compared with controls. Testosterone in boars fed altrenogest for 9 wk never exceeded 1 ng/ml. There were significant ( $P < .001$ ) treatment differences as well as treatment x week interactions ( $P < .001$ ) for testosterone concentrations in serum. Because this study was designed to be terminated when boars reached 24 wk of age or near market weight, changes in testosterone were not obtained from the 9-wk boars during altrenogest withdrawal. Concentrations of testosterone and estradiol-17 $\beta$  in serum from a spermatic cord vein at castration were decreased ( $P < .05$ ) by altrenogest treatment (table 2). However, there were no differences for either steroid among groups fed altrenogest.

## DISCUSSION

Our data demonstrate that feeding a synthetic progestogen (altrenogest) for 3, 6, or 9 wk will prevent the rise in testosterone and estradiol (Allrich et al., 1982) associated with sexual development in growing boars. Increasing concentrations of testosterone in serum were observed from about 16 to 24 wk for control boars (figure 1). This increase was delayed by feeding altrenogest for 3 or 6 wk and was inhibited completely in boars fed altrenogest continuously for 9 wk. Testosterone concentrations for all treated groups paralleled each other, but at different magnitudes during treatment (figure 1). We suggest that apparent differences in testosterone at 12 and 14 wk age (pretreatment) probably were related to differences in stage of sexual development, even though effects of age and litter were randomized when boars were assigned to treatment groups.

Our data show that the rise in testosterone for control boars began at approximately 112 to 119 d of age (figure 1). These results agree with those of Andresen (1976) who found that increases in testosterone occurred at 110 to 125 d of age. Furthermore, Allrich et al. (1982) observed that boars produced increasing amounts of testosterone and estradiol-17 $\beta$  from 130 to 160 d of age and the resulting increases in steroid concentrations in serum resulted from greater testicular sensitivity to LH (Allrich et al., 1983).

Treatment with altrenogest inhibited testicular growth (as measured by weight and volume) compared with controls. These results as well as decreased testosterone in serum are consistent with observations made by Busch et al. (1979) using CAP, another synthetic progestogen. Duration of feeding altrenogest showed a linear negative effect for these two traits. Both testicular characteristics as well as testosterone are dependent upon anterior pituitary gonadotropins (Allrich et al., 1983), and this would support the proposed suppressive action of progestogen on pituitary gonadotropin secretion (Webel, 1978).



The steroidal hormone concentrations in a spermatic cord vein were much lower than expected. It was anticipated that testosterone from testicular venous samples would be greater than peripheral concentrations. Only controls and the boars fed altrenogest for 9 wk had testicular venous testosterone concentrations that were greater numerically than peripheral levels at 24 wk of age. While magnitude of testicular venous testosterone concentrations in our control boars were about one-third of those for anesthetized boars reported by Setchell et al. (1983), testicular estrogen was nearly identical in both studies for control boars of similar ages. It is possible that the tranquilizer we administered approximately 20 min before anesthesia decreased hypothalamic LHRH secretion, thereby causing decreased LH and steroid concentrations. Alternatively, there may have been a direct negative effect of the tranquilizer on testicular steroidogenesis.

Duration of altrenogest feeding had no apparent effect on gain, feed intake, or final weight at 24 wk of age. Differences in feed conversion may have occurred due to slight nonsignificant differences in feed intake and gain, because neither trait alone was different among treatments. The fact that final weights were similar despite marked differences in testosterone may agree with earlier work. If boars with low testosterone (9-wk group) are similar to castrated males (no testicular testosterone secretion), then one would expect the low testosterone group to grow faster than the intact male under ad libitum feeding (Kay and Houseman, 1975). While similar research concluded that differences in gain between intact and castrate male pigs were not great (Teague et al., 1964; Tribble et al., 1965), other work indicated that intact males grew faster with improved feed conversion than either castrate males or gilts at higher levels of dietary protein (Speer et al., 1957; Prescott and Lamming, 1964). These responses suggest a sex x dietary protein interaction as evidenced by superior growth of boars compared with barrows when fed an 18% protein (.86% lysine) diet (Williams et al., 1984). Our diet was formulated to contain 18% protein

(.95% lysine) in the present study.

One difference observed in castrated and intact male pigs for growth and nutrient partitioning is that intact males have more muscling and leanness of carcass than castrates of comparable body weight (Prescott and Lamming, 1964; Desmoulin and Bonneau, 1979). Boars fed altrenogest (particularly those in the 9-wk group) had low testosterone in serum, but were as lean as controls. This observation may suggest that altrenogest had enough anabolic activity to partition nutrients into lean growth rather than fat deposition. However, altrenogest may not have reduced sufficiently testosterone in serum to preclude the anabolic effect of low concentrations of testosterone. Whether feeding altrenogest would suppress fat deposition in barrows is not known, but warrants further study regarding the possible nutrient partitioning of altrenogest.

There may be various threshold concentrations of testosterone in the growing boar responsible for anabolic growth, sexual behavior, libido, and maintenance of spermatogenesis and accessory gland function. Varying testosterone therapy in wethers produced sexual behavioral without mating until concentrations of testosterone were increased (D'Occhio and Brooks, 1982). Lower concentrations of testosterone in serum may be adequate for the anabolic effect characteristic of the intact male because rams and bulls grow faster than wethers and steers prior to the pubertal rise in androgens associated with testicular maturation and puberty (Seidman, 1982). If altrenogest has little or no anabolic effects, then low concentrations of testosterone in boars fed altrenogest for 9 wk were sufficient to prevent increased fat deposition compared with controls. Setchell et al. (1983) reported that anesthetized boars varying from about 60 (4 mo old) to 280 kg (adult) had higher concentrations of testosterone in spermatic venous and lymph vessels, and rete testis fluid than in arterial blood plasma. These high levels of testosterone are probably essential for spermatogenesis, but more moderate concentrations of

testosterone may be required for maintenance of the sexual accessory glands, behavior, and libido. Altrenogest suppressed high levels of testosterone normally observed in untreated boars, while possibly maintaining sufficient testosterone concentrations for optimal growth and leaner carcass composition.

Our results suggest that feeding altrenogest to growing boars will: 1) delay or inhibit sexual development by preventing the normal rise in testosterone that occurs during testicular development, 2) inhibit testicular development, and 3) have no effect on the body growth rate from 15 to 24 wk of age. This work indicates that boars could be fed altrenogest without affecting growth rate or backfat thickness. The possibility exists that intact males could be marketed with little or no boar "taint" due to the inhibition of testicular development and steroidogenesis. This supposition is based upon the accepted role of testicular steroids as precursors for the primary compound causing boar taint (5 $\alpha$ -androst-16-en-3-one) (Patterson, 1968; Andresen, 1975). Further study to determine the effects of a progestogen-induced delay of sexual development, as well as more data confirming these results, is needed.

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TABLE 1. GROWTH AND TESTICULAR CHARACTERISTICS OF BOARS FED ALTRENOGEST (20 MG/DAY) FOR 0, 3, 6, OR 9 WEEKS BEGINNING AT 15 WEEKS OF AGE AND FOLLOWING CASTRATION AT 24 WEEKS OF AGE

| Item                                    | Control | 3                | 6                | 9                | Pooled SE |
|---|---------|------------------|------------------|------------------|-----------|
| No. boars                               | 6       | 6                | 6                | 6                |           |
| Initial wt <sup>a</sup> , kg            | 43.1    | 43.3             | 44.5             | 45.2             | 1.8       |
| Final wt, kg                            | 90.9    | 93.5             | 89.9             | 96.5             | 3.9       |
| Avg backfat thickness <sup>b</sup> , cm | 1.9     | 2.0              | 1.9              | 2.1              | .1        |
| Avg daily intake, kg                    | 2.3     | 2.3              | 2.5              | 2.7              | .2        |
| Avg daily gain, g                       | 798     | 837              | 757              | 857              | 45        |
| Feed/gain                               | 3.0     | 2.8              | 3.3 <sup>e</sup> | 3.2              | .06       |
| Testicular vol <sup>cd</sup> , ml       | 448     | 348              | 277              | 189              | 26        |
| Testicular wt <sup>d</sup> , g          | 481     | 382              | 291              | 208              | 28        |
| % of total body wt                      | .53     | .40 <sup>e</sup> | .32 <sup>e</sup> | .22 <sup>e</sup> | .03       |

<sup>a</sup>Litter effect ( $P < .05$ ).

<sup>b</sup>Ultrasonically determined at 24 wk of age prior to final weight.

<sup>c</sup>Measured by water displacement.

<sup>d</sup>Linear decrease ( $P < .001$ ).

<sup>e</sup>Different from control ( $P < .05$ ).



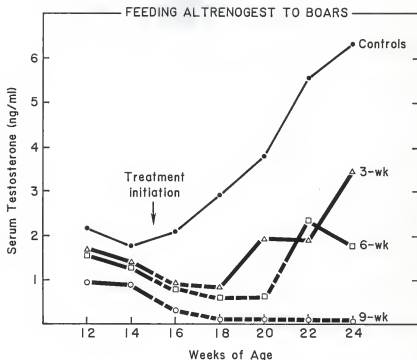


Figure 1. Serum testosterone concentrations for boars fed altrenogest (20 mg/d) for either 0, 3, 6, or 9 wk beginning at 15 wk of age. Blood was collected at biweekly intervals. Broken portion of lines indicates period of altrenogest treatment.

TABLE 2. TESTOSTERONE (NG/ML) AND ESTRADIOL (PG/ML) CONCENTRATIONS IN TESTICULAR VENOUS SERUM AT 24 WEEKS OF AGE AFTER 0, 3, 6, OR 9 WEEKS OF ALTRENOGEST FEEDING BEGINNING AT 15 WEEKS OF AGE

| Hormone                   | Control | 3    | 6   | 9   | Pooled SE |
|---------------------------|---------|------|-----|-----|-----------|
| Testosterone <sup>a</sup> | 8.7     | 2.4  | 1.2 | .3  | 1.6       |
| Estradiol <sup>a</sup>    | 119.8   | 29.3 | 8.8 | 5.0 | 30.6      |

<sup>a</sup>Control vs others ( $P < .01$ ).

GROWTH, CARCASS TRAITS, BOAR ODOR AND TESTOSTERONE IN SERUM OF  
MALE PIGS FED A PROGESTOGEN, ALTRENOGEST

ABSTRACT

Altrenogest was fed to crossbred (Chester White X Yorkshire X Duroc) boars to determine effects on body growth, carcass composition, incidence of boar odor, and testosterone in serum. Boars ( $n=54$ ) from 18 litters (block) were assigned at 12 wk of age to three treatments: 1) 18 control boars, 2) 18 boars fed altrenogest (20 mg/d) for 6 wk from 15 to 21 wk of age, followed by 4 wk with no treatment, and 3) 18 boars castrated at 2 wk of age (barrows). Boars fed altrenogest had greater ( $P<.05$ ) average daily gain (ADG) than barrows during treatment. Feed intake and ADG were lower ( $P<.05$ ) for altrenogest-treated boars than for control boars and barrows during the 4-wk withdrawal period. Daily observations of boars fed altrenogest indicated marked mounting activity beginning 5 d after withdrawal of altrenogest, possibly explaining the poor rate of growth following withdrawal of altrenogest. Boars fed altrenogest weighed less ( $P<.05$ ) than control boars and barrows at 25 wk of age (at slaughter). Both groups of boars were similar in percentage muscle and had less ( $P<.05$ ) backfat than barrows, whereas control boars had the largest ( $P<.05$ ) loin-eye areas. Concentration of testosterone in serum was decreased ( $\leq 1$  ng/ml) in boars fed altrenogest during treatment, but rose to concentrations similar to control boars at 2 and 4 wk after withdrawal of altrenogest. Intensity of boar odor in fat samples was similar in both groups of boars and was greater ( $P<.05$ ) than that for barrows based on evaluations by a trained sensory panel. Feeding altrenogest to growing boars had no negative effects on

carcass qualitative traits, or on growth performance during treatment, even though ADG was reduced after withdrawal of altrenogest. It is possible that boar odor was prevented during altrenogest treatment, but evolved rapidly in association with increased secretion of testosterone after altrenogest treatment was terminated.

(Key Words: Boar, Progestogen, Carcass, Boar Odor, Growth, Testosterone.)

## INTRODUCTION

Altrenogest, a synthetic progestogen, inhibited testicular growth and reduced testosterone in serum of growing boars, but did not alter growth or backfat thickness (Kluber et al., 1985). Chloramadinone acetate (another progestogen) fed for 70 d (30 mg/d) produced a desexualizing effect and reduced  $\Delta$ alpha-androstenone in meat and fat of boars (Busch et al., 1979).

Testicular characteristics, secretion of testosterone, and possibly boar odor are dependent upon gonadotropins (Andresen, 1975; Allrich et al., 1983), supporting the proposed suppressive action of progestogens on gonadotropin secretion (Webel, 1978). Altrenogest, in fact, might produce a castration-like effect in the growing boar by reducing testicular secretion of testosterone through suppression of LH secretion without altering advantages of boars for muscle growth. The objective of this study was to confirm earlier results of the absence of effects of altrenogest on body growth in boars, as well as to determine its effects on carcass composition and the incidence of boar odor.

## METHODS AND MATERIALS

### Animals

Crossbred male pigs (Chester White X Yorkshire X Duroc) born in March 1985 were selected from litters that consisted of both female pigs and at least five male

pigs. Two male pigs from each litter were selected randomly and castrated at approximately 2 wk of age. All pigs were weaned at 20 to 29 d of age and placed in an environmentally controlled nursery. A barley (IFN 4-00-549)-soybean meal (IFN 5-04-612) diet (20% protein; 1.25% lysine) was provided ad libitum. Starting at 9 wk of age and continuing for the remainder of the study, all pigs received a medicated (ASP-250), 18% protein (.95% lysine), milo (IFN 4-04-444)-soybean meal diet fortified with vitamins and minerals that met or exceeded all NRC (1979) recommendations. At 12 wk of age, three littermate male pigs (two boars and one barrow) of similar weights were selected from 18 litters (n=54) and were blocked by litter with one pig from each litter assigned randomly to three treatment groups. These pigs then were moved into a modified open-front facility with solid concrete floors.

#### Experimental Design

Male pigs were separated arbitrarily into heavy-, medium-, and light-weight groups and assigned randomly within treatment to pens (4.6 x 1.2 m) with three pigs per pen and six pens per treatment for the duration of the study. Treatments included: 1) control boars, 2) boars fed altrenogest for 6 wk from 15 to 21 wk of age, and 3) barrows. Feed and water were available at all times during the study. Altrenogest was mixed into the feed for the 6-wk treatment period and was provided at  $20 \text{ mg} \cdot \text{boar}^{-1} \cdot \text{d}^{-1}$  based on the calculated daily consumption of feed during the previous week. The 20-mg dose was chosen because a similar dose was adequate to reduce testosterone in serum and inhibit testicular growth in a previous study (Kluber et al., 1985). During the withdrawal period (22 to 25 wk of age) when no altrenogest was fed, altrenogest-treated boars were fed the same diet as control boars and barrows. A 30-d withdrawal period was required before slaughter to comply with regulations of the U.S. Food and Drug Administration.

Pigs were sprinkled with water for 2 min every 15 min when the ambient

temperature exceeded 29°C. After 5 weeks, one of the control boars was removed from the study because of pneumonia that was unresponsive to chemotherapy.

### Measurements

Consumption of feed and individual body weights were assessed biweekly to determine average daily feed intake (FI), average daily gain (ADG), and feed efficiency (F/G) until 25 wk of age. Blood was collected via puncture of the anterior vena cava at biweekly intervals between 0630 and 0800 h to monitor concentrations of testosterone in serum by radioimmunoassay (Kluber et al., 1985).

Pigs from the 12 heaviest litters were chosen a priori to be slaughtered at 25 wk of age to increase the probability of having boars with significant boar odor. Pigs were slaughtered on two consecutive days (18 pigs/d) beginning at 0600 h. Carcasses were processed under federal inspection. The right submaxillary gland was collected and weighed. Hot carcass weights (HCW) were determined before the carcasses were placed in the cooler. Carcass measurements were determined 24 h after slaughter using the right side of each carcass. These included: length from the first rib to the pubic bone, backfat thickness measured at the last rib, and fat depth (FD) measured between ribs 10 and 11 laterally from the dorsal midline about three-quarters down the loin eye. Loin eye area (LEA) at the 10th rib was determined using a planimeter.

The USDA grade of each carcass (USDA, 1985) was determined and color, firmness, and exudativeness of the longissimus muscle was evaluated at the cross section of the 10th rib area. Classifications were based on a scale of one to five for color (1=pale; 2=slightly pale; 3=normal grayish pink; 4=slightly dark; 5=dark), firmness (1=soft; 2=slightly soft; 3=normal firmness; 4=slightly firm; 5=firm), and exudativeness (1=watery; 2=slightly watery; 3=normal dryness; 4=slightly dry; 5=dry) as described by Rust and Topel, (1969). Percentage muscle was calculated using the formula =  $[2.1 + (.45 \times \text{HCW}) + (5 \times \text{LEA}) - (11 \times \text{FD})] / \text{HCW} \times 100$  (Boggs and

Merkel, 1982).

### Sensory Panel

Several individuals (n=13) were screened initially for their olfactory sensitivity to boar odor in fat samples. From those individuals capable of detecting differences in intensity of boar odor, six panelists (five females, one male) were trained in a two-phase procedure. The first phase was accomplished by exposing panelists to individual samples of backfat of known intensity in two daily sessions of 15 min for 1 wk. The following week (second phase), panelists were asked to select samples of similar intensity by evaluating three samples of which two were selected from the same carcass during twice daily sessions lasting 15 min. Test samples consisted of 1-cm cubes that were excised from an 11.4 x 15.2 cm full thickness sample of backfat collected from an area cranial to the 10th rib on the left side. Test samples were heated on individual porcelain hot plates that were precalibrated to a surface temperature of 147 to 153 C. Heated surfaces were cleaned with 70% ethanol between samples. Testing sessions were conducted twice daily (0830 and 1330 h) on three consecutive days for a 2-wk period for a total of 12 sessions. Representative samples from one litter and from each treatment group (a total of seven samples) were evaluated at each session. Panelists used a scale of 1 to 5 to score the intensity of boar odor for each sample (1=none; 2=slight; 3=moderate; 4=strong; 5=very strong).

### Statistical Analyses

Data were analyzed as a randomized complete-block experiment including treatment and litter (where appropriate), using the general linear models procedure of the Statistical Analysis System (SAS, 1979). Pen was considered the experimental unit for growth, feed intake, and feed conversion. Preplanned orthogonal contrasts were used to separate treatment means (control or altrenogest-treated boars vs

barrows) and a nonorthogonal contrast (treated vs control boars) by LSD (SAS, 1979) also was made.

The sensory experiment was designed as a repeated measure, crossover design using all six possible sequences of the three treatments. Litter effects were included in the statistical model as a block effect that was confounded with panel sessions. Each panelist was assigned a sampling sequence, thus panelist and sequence effects were confounded. At each session, a sample from a control boar, a barrow, and a treated boar, all from the same litter, were each evaluated twice by each panelist. To balance carryover effects, each panelist evaluated seven samples, two from two of the treatments, and three from the third. The first of seven samples was not used leaving two samples from each treatment to be evaluated by each panelist during each session. Every treatment was preceded by each of the other treatments, but not preceded by itself. The experiment was thus balanced for carryover effects that were estimated using dummy covariates as outlined by Milliken and Johnson (1984).

## RESULTS

Concentrations of testosterone in serum collected on alternate weeks from 13 to 25 wk of age are illustrated in figure 1. Testosterone was lower ( $P<.05$ ) in the serum of boars fed altrenogest than in that of control boars 2 wk after initiation of treatment. Testosterone in serum of the boars fed altrenogest remained about 1 ng/ml for the duration of the treatment period. Testosterone returned to concentrations similar to those of control boars in the first sample collected 2 wk after withdrawal of altrenogest. Testosterone in serum of barrows never exceeded .2 ng/ml.

Growth performance was analyzed separately for the 6-wk treatment period and for the 4-wk withdrawal period (table 1). Pigs averaged  $51.6 \pm 1.5$  kg at the



beginning of the study with the barrows being heavier ( $P<.05$ ) than the control boars. Control boars consumed less ( $P<.05$ ) feed than barrows and boars receiving altrenogest during the treatment period. During the withdrawal period, both control and altrenogest boars consumed less ( $P<.05$ ) feed than barrows.

Average daily gain based on biweekly measurements from 15 to 25 wk of age is illustrated in figure 2. During the 6-wk treatment period, boars fed altrenogest had greater ( $P<.05$ ) ADG than barrows and a tendency for greater ( $P=.08$ ) ADG than control boars, even though there was no difference in live weight between treatment groups at 21 wk of age (table 1). After withdrawal, ADG of boars fed altrenogest decreased markedly (figure 2). Previous treatment with altrenogest was associated with a 56% reduction ( $P<.05$ ) in ADG and a 44% reduction in daily feed intake during withdrawal (table 1). During the first 2-wk period of withdrawal, some boars previously fed altrenogest lost weight. Control boars and barrows gained at similar rates during this 4-wk period.

Feed efficiency was similar for the altrenogest-fed boars and barrows during the treatment period, whereas control boars were more ( $P<.05$ ) feed efficient than either group. During the withdrawal period, control boars were more ( $P<.05$ ) feed efficient than barrows, and treated boars were less ( $P<.05$ ) efficient than both groups.

Overall, treated and control boars consumed less ( $P<.05$ ) feed than barrows from 15 to 25 wk of age, whereas control boars and barrows had similar ADG. Altrenogest-treated boars were lighter ( $P<.05$ ) than boars and barrows at 25 wk of age (table 1). This resulted in similar feed efficiencies for barrows and altrenogest-treated boars, whereas control boars were most ( $P<.05$ ) efficient.

Pigs from the 12 heaviest litters were slaughtered after a 32-d withdrawal period for determination of carcass characteristics (table 2). Slaughter weight for the

altrenogest-treated boars was less ( $P<.05$ ) than that for control boars and barrows, but carcass dressing percentages were similar among treatment groups. Boars fed altrenogest had lighter ( $P<.05$ ) HCW than control boars and barrows. Control boars had longer ( $P<.05$ ) carcasses than the other two groups.

Carcass fat and muscle traits were determined 24 h after slaughter (table 3). Both control and altrenogest-treated boars had less ( $P<.05$ ) backfat over the last rib and higher ( $P<.05$ ) percentage of muscle than barrows. Control boars had larger ( $P<.05$ ) loin-eye areas than barrows. Both boar groups had lower ( $P<.05$ ) USDA grade numbers than barrows because they had leaner, more muscular carcasses. Treatment groups were similar for color, firmness, and exudativeness of the longissimus muscle at the 10th rib.

Intensity of boar odor was similar between the control and altrenogest-treated boars. Mean scores for intensity of boar odor ranged from slight to moderate in both boar groups, and were higher ( $P<.05$ ) than those for barrows. Of the 36 pigs evaluated, only seven (five control boars and two altrenogest-treated boars) had intensity scores that averaged 3.0 (moderate) or higher. Only one altrenogest-treated boar scored greater than one standard deviation (1.2) above the treatment mean of 2.5.

Weight of the submaxillary glands was similar for boars fed altrenogest ( $52 \pm 3$  g) and barrows ( $47 \pm 3$  g), but both were lighter ( $P<.05$ ) than those of control boars ( $72 \pm 3$  g).

## DISCUSSION

Our data confirm that feeding altrenogest (progestogen) for 6 wk will prevent the rise of testosterone in serum associated with sexual development in growing boars (Allrich et al., 1982; Kluber et al., 1985). Increasing concentrations of

testosterone in serum were observed from 13 to 25 wk for control boars (figure 1). This increase was delayed for 6 wk by feeding altrenogest. Our data tend to support earlier work (Andresen, 1976; Kluber et al., 1985) suggesting that the pubertal rise in testosterone for control boars begins at approximately 16 to 17 wk of age, even though testosterone for our control boars began to increase around 15 wk of age. This earlier rise of testosterone in serum might be associated with earlier pubertal development in our three breed rotation, resulting from heterosis of reproductive traits in peripubertal boars (Neely et al., 1980).

Treatment with altrenogest did not appear to alter ADG during the treatment period. These data concur with our earlier findings that feeding altrenogest to growing boars did not alter growth rate (Kluber et al., 1985). However, we observed a tendency ( $P=.08$ ) for boars fed altrenogest to grow faster than control boars during the treatment period. However, treated boars consumed more feed and were less feed efficient than control boars when compared with those in our earlier experiment where no differences in ADG or feed efficiency were observed.

During the withdrawal period, growth performance of altrenogest-treated boars was reduced compared with control boars and barrows. The decline in ADG between 22 and 25 wk for control boars and barrows might have been due to behavioral characteristics associated with increased concentrations of testosterone in serum of treated boars after withdrawal of altrenogest (figures 1 and 2). Considerable mounting activity by the treated boars as early as 5 d after withdrawal of altrenogest was observed during daily visits to the facility to care for the pigs. The mounting activity of boars previously fed altrenogest persisted throughout the remainder of the study. Some mounting of control boars was observed, but not as much as observed for the treated boars. Increased mounting activity of peripubertal boars occurred between 16 and 18 wk of age in earlier studies (Tonn et al., 1985)

and coincides with increasing concentrations of testosterone in serum of our boars (figure 1), as well as in other studies (Colenbrander, 1978; FlorCruz and Lapwood, 1978). Because altrenogest-treated boars were housed between pens of control boars and barrows, feeding behavior of all pigs may have been disrupted from 21 to 23 wk of age as evidenced by a decline in ADG for all treatment groups (figure 2). Boars previously fed altrenogest had reduced feed intake (table 1) and marked reduction in ADG during wk 21 to 23 (figure 2). We did not observe obvious changes in such mounting activity in our previous study (Kluber et al., 1985), nor was feed intake reduced after withdrawal of altrenogest in that study. The mounting observed after the withdrawal of altrenogest in our present study was probably related to the rise in testosterone. This could be expected based on the increased mounting activity that is normally observed when testosterone is increasing in the serum of peripubertal boars (Colenbrander, 1978; FlorCruz and Lapwood, 1978; Tonn et al., 1985). Testosterone in serum increased to concentrations similar to those of control boars within 2 wk of treatment withdrawal. Indeed, testosterone may have increased more rapidly, but that could not be determined by the sampling frequency we utilized. In contrast to our observations, Busch et al. (1979) did not report increased sexual activity of boars during an 80-d withdrawal period following treatment with chloramadinone acetate.

Overall, control boars and barrows had equivalent ADG, a result similar to earlier research in which differences in gain between intact and castrated male pigs were small (Teague et al., 1964; Tribble et al., 1965). One difference observed between castrated and intact male pigs was that intact males had more muscling and leaner carcasses than castrates of comparable body weight (Prescott and Lamming, 1965; Desmoulin and Bonneau, 1979). In our study, control and treated boars were comparable for muscling and leanness, and both were superior to barrows. Our

previous work suggested that altrenogest treatment in boars did not alter growth rate, even though testosterone in serum was reduced (Kluber et al., 1985). Our present study further suggested that lower concentrations of testosterone in serum of boars fed altrenogest were sufficient to partition nutrients into lean growth rather than fat deposition. Alternatively, it is possible that altrenogest had slight anabolic activity in the boar to partition nutrients into more lean growth in the face of low (barrow-like) concentrations of testosterone. However, because altrenogest administered subcutaneously shows weak anabolic and androgenic activity equivalent to about 1/20 of that of testosterone (Personal communication, Roussel-UCLAF), it might be suggested that the anabolic effects observed in the intact boar requires very low concentrations of testosterone in serum, but more than that observed in the barrow (figure 1).

Testicular steroids are precursors for the primary compound causing boar odor (5 $\alpha$ -androst-16-en-3-one, Patterson, 1968; Andresen, 1975; Brooks and Pearson, 1986). Submaxillary salivary glands metabolize steroid precursors to the 5 $\alpha$ -androst-16-en-3-one products responsible for boar odor (Brooks and Pearson, 1986). Even though altrenogest reduced the weight of the submaxillary glands in comparison to control boars, there was no difference in the intensity of boar odor 4 wk after withdrawal of altrenogest from the diet. We hypothesize that intensity of boar odor would have been lower at the end of the altrenogest-feeding period. Our supposition may have merit because Booth (1975) found that concentrations of 5 $\alpha$ -androst-16-en-3-one did not increase until after 24 wk of age in boars. We propose that boar odor increased in parallel to increased testosterone in serum during the 32-d period after withdrawal of altrenogest. Alternatively, concentrations of testosterone in serum of approximately 1ng/ml may have indicated adequate steroidogenic activity in the testes to provide boar-odor steroids equivalent to

control boars.

Our results suggest that feeding altrenogest (progestogen) to growing boars may: 1) slightly increase body growth; 2) have no adverse effects on muscle quality; and 3) have little or no effect on muscling or leanness of the carcass when compared to control boars. In addition, intensity of boar odor may not be reduced if altrenogest treatment is terminated several weeks before slaughter. It appears that intact males possibly could be marketed with little or no boar odor if testicular secretion of steroids was reduced by feeding altrenogest or administering another progestogen until slaughter. This could be accomplished while maintaining muscling and leanness characteristics similar to those of intact boars. We believe our results are sufficiently encouraging to warrant further testing of other progestogen treatment regimens that avoid abrupt withdrawal of progestogen before slaughter. In this regard, an implant might be advantageous for providing a slow withdrawal from progestogen treatment and avoiding a possible acute rise of testosterone in serum. However, further study to determine the reduction in boar odor from carcass fat by treating with progestogens until slaughter are required before these possibilities can be evaluated.

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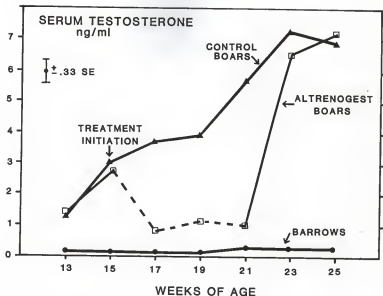


Figure 1. Concentrations of testosterone in serum of boars fed altrenogest (20 mg/d) for 6 wk beginning at 15 wk of age. Blood was collected at biweekly intervals. Broken portion of line indicates period of altrenogest treatment.

TABLE 1. BODY WEIGHT, AVERAGE DAILY GAIN, DAILY FEED INTAKE, AND FEED EFFICIENCY DURING ALTRENOGEST TREATMENT AND WITHDRAWAL PERIODS

| Item                        | Age, wk | Control boars      | Altrenogest <sup>a</sup> boars | Barrows            | SE  |
|-----------------------------|---------|--------------------|--------------------------------|--------------------|-----|
| Initial body wt, kg         |         | 50.4 <sup>b</sup>  | 51.2 <sup>bc</sup>             | 53.4 <sup>c</sup>  | .9  |
| Treatment period            | 15-21   |                    |                                |                    |     |
| FI, kg                      |         | 2.74 <sup>b</sup>  | 3.51 <sup>c</sup>              | 3.29 <sup>c</sup>  | .12 |
| ADG, kg                     |         | .93 <sup>bc</sup>  | .99 <sup>b</sup>               | .88 <sup>c</sup>   | .02 |
| F/G                         |         | 2.92 <sup>b</sup>  | 3.57 <sup>c</sup>              | 3.74 <sup>c</sup>  | .13 |
| Body wt after treatment, kg |         | 89.8               | 92.6                           | 90.5               | 1.3 |
| Withdrawal period           | 21-25   |                    |                                |                    |     |
| FI, kg                      |         | 2.98 <sup>b</sup>  | 1.97 <sup>c</sup>              | 3.38 <sup>d</sup>  | .08 |
| ADG, kg                     |         | .85 <sup>b</sup>   | .44 <sup>c</sup>               | .82 <sup>b</sup>   | .03 |
| F/G                         |         | 3.52 <sup>b</sup>  | 4.64 <sup>c</sup>              | 4.11 <sup>d</sup>  | .19 |
| Final body wt, kg           |         | 113.6 <sup>b</sup> | 104.9 <sup>c</sup>             | 113.6 <sup>b</sup> | 1.4 |
| Overall                     | 15-25   |                    |                                |                    |     |
| FI, kg                      |         | 2.83 <sup>b</sup>  | 2.89 <sup>b</sup>              | 3.33 <sup>c</sup>  | .08 |
| ADG, kg                     |         | .90 <sup>c</sup>   | .77 <sup>b</sup>               | .86 <sup>c</sup>   | .03 |
| F/G                         |         | 3.13 <sup>b</sup>  | 3.79 <sup>c</sup>              | 3.88 <sup>c</sup>  | .11 |

<sup>a</sup>Daily dose (20 mg/d) from 15 to 21 wk of age.

<sup>b,c,d</sup>Means with unlike superscripts are different ( $P < .05$ ).

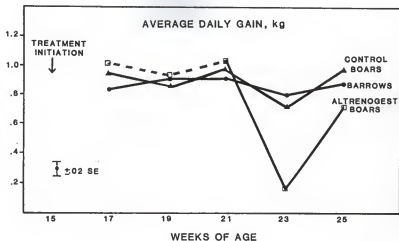


Figure 2. Average daily gain of boars fed altrenogest (20 mg/d) for 6 wk beginning at 15 wk of age. Male pigs were weighed biweekly with the first biweekly period ending at 17 wk of age.

TABLE 2. CARCASS CHARACTERISTICS AT 25 WEEKS OF AGE

| Item               | Control<br>boars   | Altrenogest<br>boars | Barrows            | SE  |
|--------------------|--------------------|----------------------|--------------------|-----|
| Slaughter wt, kg   | 117.8 <sup>a</sup> | 110.1 <sup>b</sup>   | 115.1 <sup>a</sup> | 1.8 |
| Hot carcass wt, kg | 85.9 <sup>a</sup>  | 80.8 <sup>b</sup>    | 85.9 <sup>a</sup>  | 1.7 |
| % Dressing         | 73.0               | 73.4                 | 74.5               | .68 |
| Length, cm         | 85.8 <sup>a</sup>  | 84.3 <sup>b</sup>    | 83.3 <sup>b</sup>  | .5  |

<sup>a,b</sup>Means with unlike superscripts were different ( $P<.05$ ).

TABLE 3. CARCASS FAT AND MUSCLE TRAITS AT SLAUGHTER (25 WEEKS OF AGE)

| Item  | Control<br>boars  | Altrenogest<br>boars | Barrows           | SE  |
|---|-------------------|----------------------|-------------------|-----|
| Loineye area <sup>a</sup> , cm <sup>2</sup> | 37.0 <sup>c</sup> | 33.6 <sup>cd</sup>   | 31.5 <sup>d</sup> | 1.4 |
| % muscle                                    | 56.1 <sup>c</sup> | 54.8 <sup>c</sup>    | 51.7 <sup>d</sup> | .9  |
| Fat thickness <sup>b</sup> , cm             | 2.40 <sup>c</sup> | 2.54 <sup>c</sup>    | 2.95 <sup>d</sup> | .1  |
| USDA grade                                  | 1.3 <sup>c</sup>  | 1.6 <sup>c</sup>     | 2.2 <sup>d</sup>  | .2  |
| Color <sup>a</sup>                          | 2.7               | 2.7                  | 2.5               | .2  |
| Firmness <sup>a</sup>                       | 2.7               | 2.5                  | 2.5               | .1  |
| Exudativeness <sup>a</sup>                  | 2.8               | 2.7                  | 2.6               | .1  |
| Boar odor intensity                         | 2.7 <sup>c</sup>  | 2.5 <sup>c</sup>     | 1.8 <sup>d</sup>  | .2  |

<sup>a</sup>Measured at the 10th rib.<sup>b</sup>Measured at the last rib.<sup>c,d</sup>Means with unlike superscripts are different (P<.05).

## ALTRENOGEST AND REPRODUCTIVE DEVELOPMENT IN BOARS

## ABSTRACT

Crossbred male pigs (n=54, Yorkshire x Duroc x Chester White) were used to evaluate the influence of feeding altrenogest (a synthetic progestogen) on reproductive development. Treatments consisted of littermate male pigs: 1) control boars (n=18), 2) boars fed daily 20 mg altrenogest (n=18) from 15 to 21 wk of age, and 3) barrows (n=18) previously castrated at 2 wk of age. Blood was collected biweekly from 13 to 33 wk of age and concentrations of testosterone and luteinizing hormone (LH) in serum were determined. Pigs from the twelve heaviest litters were sacrificed at 25 wk of age (4 wk after altrenogest withdrawal) and the remaining control boars (n=5) and altrenogest-treated boars (n=6) were castrated at 33 wk of age (12 wk after altrenogest withdrawal). Body of the prostate, paired seminal vesicles, and paired bulbourethral glands of altrenogest-treated boars weighed less ( $P<0.05$ ) than those of control boars, but were heavier ( $P<0.05$ ) for altrenogest boars than for barrows. Weights of the paired testes and epididymides and content of sperm were less ( $P<0.05$ ) for altrenogest than control boars at 25 wk of age, but were similar at 33 wk of age. Total seminiferous epithelium and total interstitium per testis were less ( $P<0.05$ ) in altrenogest than control boars 4 wk after withdrawal of altrenogest, but by 33 wk of age, total seminiferous epithelium and total interstitium per testis were similar for both groups of boars. Total tubular diameter, luminal diameter, and epithelial height of seminiferous tubules at Stages 1 and 8 were similar for both groups of boars at both ages. Concentrations of testosterone and LH in serum were similar for all boars prior to

treatment with altrenogest. Concentrations of LH and testosterone were reduced ( $P<0.05$ ) for altrenogest boars during treatment, but increased ( $P<0.05$ ) within 2 wk after withdrawal of altrenogest and were similar in all boars from 23 to 33 wk of age. Testicular parenchyma of boars previously fed altrenogest produced less ( $P<0.05$ ) testosterone and estradiol-17 $\beta$  in vitro in the absence and presence of human chorionic gonadotropin than control boars at 33 wk of age. These data suggest that feeding altrenogest to peripubertal boars delayed puberty, but did not reduce permanently endocrine and testicular functions.

### INTRODUCTION

Progesterone has been shown to reduce testosterone-dependent male sexual behavior in ring doves (Erickson et al., 1967), pigeons (Murton et al., 1969), guinea pigs (Diamond, 1966), mice (Erpino, 1973), and gerbils (Griffo and Lee, 1973). Busch et al. (1979) found that chloramadinone acetate (synthetic progestogen) fed for 70 d produced a desexualizing effect on boars. We observed that feeding boars another progestogen, altrenogest: 1) delays or inhibits sexual development by preventing the normal rise of testosterone in serum that occurs during reproductive development, 2) inhibits testicular development, and 3) has no effect on rate of body growth from 15 to 24 wk of age (Kluber et al., 1985). However, the mode by which altrenogest reduces testicular development and its long-term effects on reproductive and endocrine function in boars have not been evaluated. The objective of this study was to determine if feeding altrenogest merely delays puberty or permanently alters testicular function in boars fed the progestogen during reproductive development.

### METHODS AND MATERIALS

#### Experimental Design

Crossbred male pigs (Chester White x Yorkshire x Duroc) born in March,



1985 were selected from litters that consisted of five or more male pigs. Two boars from each litter were selected randomly and castrated at approximately 2 wk of age. All pigs were weaned at approximately 3 wk of age into an environmentally controlled nursery. A pelleted complete diet (18% protein; 1.25% lysine) was provided *ad libitum* for 2 wk. Then, a medicated (ASP-250), 18% protein (.95% lysine) milo-soybean meal diet fortified with vitamins and minerals, that met or exceeded all National Research Council (1979) recommendations, was provided for the remainder of the study.

At 12 wk of age, three littermates ( $n=54$ , two intact boars and one barrow) of similar weights were selected from 18 litters (block) with one pig from each litter assigned randomly to each of three treatments and moved into a modified open-front facility with solid concrete floors. Male pigs were separated into arbitrary heavy-, medium-, and light-weight groups and assigned randomly within treatment to similar pens (4.6 x 1.2 m). There were three pigs/pen and six pens/treatment for the duration of the study. Treatment groups included: 1) control intact boars, 2) intact boars fed altrenogest (20 mg/d) for 6 wk from 15 to 21 wk of age, and 3) barrows castrated at 2 wk of age. The 20-mg dose was chosen because a similar dose was adequate to suppress testosterone in serum of prepubertal boars (Kluber et al., 1985).

Pigs were provided feed and water at all times. Altrenogest was mixed into the feed during the treatment period and was provided at  $20 \text{ mg} \cdot \text{boar}^{-1} \cdot \text{d}^{-1}$  based on the estimated daily feed consumption during the previous 1-wk period. During the withdrawal period (22 to 25 wk of age) when no altrenogest was fed, altrenogest-treated boars were fed the same diet as control boars and barrows. A 30-d withdrawal period was required before slaughter to comply with regulations of the U.S. Food and Drug Administration.

Pigs were sprinkled with water for 2 min every 15 min when the ambient temperature exceeded 29°C. After 5 weeks, one of the control boars was removed from the study because of pneumonia that was unresponsive to chemotherapy.

#### Measurements

Blood was collected via puncture of the anterior vena cava at biweekly intervals from 13 to 33 wk of age. All samples were collected between 0630 and 0900 h and placed on ice. Serum was separated and frozen until assayed for testosterone and luteinizing hormone (LH).

Pigs from the twelve heaviest litters (12/treatment) were chosen a priori to be sacrificed at 25 wk of age on two consecutive days (18 pigs/d) beginning at 0600 h. Carcasses were processed under federal inspection. Several hairs were collected from the dorsal midline of the neck approximately 12 cm caudal to the poll to serve as a bioassay for "maleness". Average diameter was calculated from measurements of the base of five hairs using a microscope with an eyepiece micrometer. Testes, epididymides, and the accessory reproductive glands (body of prostate, bulbourethral glands, and seminal vesicles) were collected, trimmed, and weighed following slaughter.

The remaining control boars (n=5) and altrenogest-treated boars (n=6) were not sacrificed, but were maintained to 33 wk of age to evaluate long-term effects of previous treatment with altrenogest on testicular function. These boars were limit-fed 3.6 kg of the same diet until castrated bilaterally at 33 wk of age. Boars were anesthetized with 1 to 1.7 g sodium thiamylal and testes and epididymides were removed surgically and weighed. Hair samples from the dorsal midline 12 cm caudal to the poll also were collected.

At castration, portions of the left testis were fixed in Bouin's solution and 6- $\mu$ m sections were prepared. Every tenth section was mounted on a glass slide and

stained with Harris' hematoxylin (Lillie, 1954). Histological examinations were similar to those described by Barnes et al. (1980). The right testis and epididymis of each boar at slaughter and castration were frozen, and later homogenized to quantitate content of sperm (Amann and Lambiase, 1969).

#### Testicular Incubations

Within 15 min after castration (33 wk of age), parenchymal tissue from the left testis was dissected free and cut into 500-mg pieces for incubation (Allrich et al., 1983). Tissue was incubated for 1 h in duplicate 25-ml Erlenmeyer flasks with 5 ml of TC 199 (25 mM Hepes) containing 0 or 1,000 IU human chorionic gonadotropin (hCG)/ml (Sigma Chemical Co., St. Louis, MO). Samples of media were frozen until assayed for testosterone and estradiol-17 $\beta$ .

#### Radioimmunoassays

Concentrations of LH in porcine serum (pLH) were determined by a double-antibody radioimmunoassay similar to that described by Kraeling et al. (1982) with modifications. Purified porcine LH (pLH) (USDA-pLH-I-1, 2.5  $\mu$ g) was reacted with 15  $\mu$ g chloramine-T and 500  $\mu$ Ci  $^{125}$ I. The reaction was stopped with 60  $\mu$ g sodium metabisulfite and  $^{125}$ I-pLH was separated from free  $^{125}$ I by anion exchange (AG 2x8, chloride form, 100-200 mesh, BioRad Laboratories, Richmond, CA) and gel filtration chromatography (Bio-Gel P-60, 100-200 mesh, BioRad Laboratories, Richmond, CA). For the assay, a large pool of porcine serum was filtered through a hollow fiber filter system (Amicon Corp., Danvers, MA) to remove pituitary hormones (exclusion limit  $\geq$  10,000 MW). Albumin from chicken eggs (Sigma Chemical Co., St. Louis, MO) was added to the pool of filtered serum to give a 5% (w/v) solution (EA-FPS). Standard curves were prepared in EA-FPS ranging from 0.05 to 10 ng USDA-pLH-I-1/200  $\mu$ l EA-FPS. Binding of  $^{125}$ I-pLH to antisera was similar for tubes containing 200  $\mu$ l EA-FPS plus 300  $\mu$ l assay buffer

(0.1 M phosphate buffered saline with 1% bovine serum albumin, pH 7.5) and for tubes with 500  $\mu$ l assay buffer. The antiserum (Chemicon International, Inc., El Segundo, CA) did not crossreact significantly with USDA-pGH-B-1 (2.1%), USDA-pFSH-B-1 (0.2%) or USDA-pPRL-B-1 (<0.06%). Increasing volumes of barrow serum displaced  $^{125}$ I-pLH from the antisera to produce a binding curve that was parallel to the standard curve. When 0.3, 0.6, 1.2, 2.5, 5.0 and 10.0 ng USDA-pLH-1-1/ml were added to EA-FPS, 0.3, 0.4, 1.2, 2.4, 5.9 and 12.4 ng were recovered (average 99.2% recovery). Sensitivity of the assay was .062 ng per assay tube. All samples were quantitated in a single assay and the within assay coefficient of variation was 17% for a pool of porcine serum averaging 0.8 ng/ml.

Concentrations of testosterone and estradiol-17 $\beta$  in serum were measured by radioimmunoassays previously validated in our laboratory (Kluber et al., 1985). Both assays were revalidated to quantitate steroids in culture media (TC 199). When 50, 100, 150 and 200 pg estradiol-17 $\beta$  was added to 100  $\mu$ l TC 199, 53, 109, 157 and 207 pg were recovered (average 105% recovery). Increasing volumes of TC 199 plus 50 pg estradiol-17 $\beta$  displaced  $^3$ H-estradiol-17 $\beta$  from the antisera to produce a binding curve that was parallel to the standard curve. Sensitivity was 5 pg/tube and the coefficient of variation of one assay was 12%. For the testosterone assay in media, recovery of added testosterone averaged 98% and variable volumes of media plus 100 pg testosterone displaced  $^3$ H-testosterone from the antisera to produce a binding curve that paralleled the standard curve. Sensitivity was 25 pg/tube and the coefficient of variation of one assay was 14%.

### Statistical Analyses

Data were analyzed as a randomized complete-block experiment including treatment and litter (where appropriate), using the general linear models procedures of the Statistical Analysis System (SAS, 1979). Concentrations of

testosterone and LH in serum were analyzed statistically as a split-plot experiment with repeated measurements (Gill and Hafs, 1971). Treatment was tested by the pig (treatment) error term. Treatment means (control or altrenogest-treated boars vs barrows) were separated using preplanned orthogonal contrasts and one nonorthogonal contrast (treated vs control boars) was made using the least-significant difference test (SAS, 1979).

## RESULTS

Growth performance, carcass traits, and boar odor for these pigs were reported elsewhere (Kluber et al., 1986). Concentrations of LH in serum collected on alternate weeks from 13 to 33 wk of age are illustrated in Fig. 1. Concentrations of LH were similar in the two groups of boars during the pretreatment period (13 to 15 wk of age). Concentrations of LH declined numerically from .9 to .7 ng/ml from 15 to 25 wk of age in control boars. As treatment with altrenogest was initiated at 15 wk of age, concentrations of LH in serum declined ( $P<0.05$ ) from 1.0 ng/ml to .3 ng/ml at 17 wk of age. This concentration was maintained until 21 wk of age when LH appeared to rise to .5 ng/ml before withdrawal of altrenogest. Thereafter, serum concentrations of LH remained unchanged until 25 wk of age. At 13 wk of age, concentrations of LH in the serum of barrows was 2.0 ng/ml and increased numerically to 2.3 ng/ml 2 wk later. From 15 to 25 wk of age, average concentrations of LH in the barrows declined to a final concentration of 1.8 ng/ml. At 27 wk of age, concentrations of LH for the remaining 11 boars (five control and six altrenogest-treated) were similar. At 29 wk of age, concentrations of LH in the control boars rose to 1.4 ng/ml and were higher ( $P<0.05$ ) than those of the altrenogest-fed boars (.7 ng/ml). At 31 and 33 wk of age, concentrations of LH were similar for all boars.

Concentrations of testosterone in serum collected on alternate weeks from

13 to 33 wk of age are illustrated in Fig. 2. Testosterone was reduced ( $P<0.05$ ) in the serum of boars fed altrenogest compared with that of control boars 2 wk after initiation of treatment. Testosterone in serum of the boars fed altrenogest remained about 1 ng/ml for the duration of the treatment period, but then increased to concentrations similar to those of control boars 2 wk after withdrawal of altrenogest and remained similar to the controls until 33 wk of age. Control boars had higher ( $P<0.05$ ) concentrations of testosterone in serum at 33 wk of age than boars fed altrenogest. As expected, testosterone in serum of barrows was low and never exceeded 0.2 ng/ml.

Weights of paired seminal vesicles, body of the prostate, and paired bulbourethral glands were lightest ( $P<0.05$ ) in the barrows (Table 1). All sexual accessory glands of the boars fed altrenogest were lighter ( $P<0.05$ ) than those of controls, but were heavier ( $P<0.05$ ) than those of the barrows.

Testicular, epididymal and body weights were reduced ( $P<0.05$ ) at 25 wk of age in boars fed altrenogest (Table 2). Total sperm per testis or epididymis and concentration of sperm/g of each tissue were less ( $P<0.05$ ) for altrenogest-treated boars than for control boars at 25 wk of age. However, at 33 wk of age, there was no difference in the weight of the testes, epididymides, or in sperm content for the control and altrenogest-treated boars.

Average diameter of hair was similar for altrenogest-treated boars and barrows (.219 and .216 mm, respectively) at 25 wk. Hair diameter tended ( $P=0.09$ ) to be greater (.237 mm) for the control boars. At 33 wk of age, average hair diameters were similar for control (.220 mm) and altrenogest-treated (.217 mm) boars.

Testicular histology of the boar groups is summarized in Table 3. Total seminiferous epithelium and total interstitium per testis were less ( $P<0.05$ ) for

boars previously fed altrenogest than for control boars 4 wk after withdrawal of altrenogest. At 12 wk after withdrawal of altrenogest (33 wk of age), total seminiferous epithelium and total interstitium per testis were similar for control and altrenogest-treated boars. Total tubular diameter, luminal diameter, and height of the seminiferous epithelium at Stages 1 and 8 were similar for all boars at both ages. Boars fed altrenogest had a greater ( $P<0.05$ ) percentage of lumen of the seminiferous tubules than control boars at 25 and 33 wk of age, whereas control boars had a greater ( $P<0.05$ ) percentage of interstitium than altrenogest-treated boars at 25 wk of age only.

Concentrations of testosterone and estradiol-17 $\beta$  from incubations of testicular parenchyma of 33-wk old boars in the presence or absence of hCG are summarized in Fig. 3. Concentrations of testosterone and estradiol-17 $\beta$  in media were lower ( $P<0.05$ ) for altrenogest-treated boars than for control boars, regardless of hCG stimulation.

## DISCUSSION

Altrenogest (progestogen) has been shown to suppress preovulatory surges of LH (Redmer and Day, 1981) and ovarian follicular development (Guthrie and Bolt, 1982) in the gilt. Our data demonstrate that feeding altrenogest for 6 wk will reduce secretion of LH and testosterone that is associated with normal sexual development of growing boars (Allrich et al., 1982). Feeding altrenogest also produced temporary castration-like effects in boars and suppressed concentrations of LH and testosterone in serum.

Leydig cells are the principle steroid-producing cells in male mammals (Christensen, 1975) and are responsive to LH (Schanbacher, 1979). Although Allrich et al. (1982) found little change in concentrations of LH in serum during peripubertal development in the boar, our data suggest that concentrations of LH

in serum decreased slightly from 13 to 23 wk of age in control boars. Treatment with altrenogest further suppressed LH (Fig. 1). This suppression was maintained until just prior to withdrawal of altrenogest. The reason for this increase in LH may be a compensatory mechanism attempting to restore suppressed concentrations of testosterone to pubertal concentrations. After withdrawal of altrenogest, LH returned to concentrations similar to control boars from 21 to 25 wk of age. Similar decreases in LH were seen in prepubertal rams treated with implants of progesterone (Echternkamp and Lunstra, 1984).

Feeding altrenogest for 6 wk prevented the rise of testosterone in serum associated with sexual development in growing boars (Kluber et al., 1985). Increasing concentrations of testosterone in serum were observed from 13 to 25 wk of age for control boars (Fig. 2). This increase was delayed for 6 wk by feeding altrenogest and corresponded to a period of lower concentrations of LH in serum. Our data support earlier work (Andresen, 1976; Kluber et al., 1985) where it was reported that the pubertal rise in testosterone for control boars began at approximately 16 to 17 wk of age.

Secretion of testosterone by Leydig cells provides a high concentration of testosterone in the seminiferous tubules that is essential for spermatogenesis (Albert, 1961), and maturation of testosterone-dependent sexual traits and growth of accessory reproductive glands (Amann and Schanbacher, 1983). Treatment with altrenogest inhibited testicular function and inhibited growth and development of accessory glands. In the testes of the boars fed altrenogest, there was less interstitium and more lumen. With reduced LH stimulation, differentiation of Leydig cells may have been delayed or inhibited compared to those of control boars (Lunstra et al., 1986). This may explain why lower concentrations of testosterone were observed in serum of treated boars and why sperm contents in



the testis and epididymis were lower at 25 wk of age than for those of control boars.

Similar effects on concentrations of LH and testosterone in serum and testicular function have been cited using chloramadinone acetate, another progestogen, and progesterone as in our present study. Busch et al. (1979) found chloramadinone acetate to have desexualizing effects in the growing boar. Concentrations of LH in serum were not measured; however, testosterone was suppressed. Testosterone-dependent characteristics including testicular weight, spermatogenesis, libido, and boar odor in fat were lower than those of control boars. But there was no difference in number of Leydig cells between control boars and treated boars. Rams treated with implants of progesterone (Echternkamp and Lunstra, 1984) had reduced testicular size, decreased spermatid maturation, and greater seminiferous tubule diameter at 14 wk of age, but by 22 wk of age (8 wk after withdrawal), there was no difference in scrotal circumference, seminiferous tubule diameter, or spermatogenesis between control and implanted rams. These observations suggest that the inhibiting effects of progesterone were largely reversible in rams as we have observed in boars in the present study.

By 33 wk of age in the present study, there were no differences in testicular size or sperm content. However, testicular tissue from boars previously fed altrenogest had reduced ability to synthesize testosterone and estradiol-17 $\beta$  in vitro compared with those of control boars, even though peripheral concentrations of testosterone in serum were quite similar (Fig. 2). Increased production of both steroids in the presence of 1,000 IU hCG did not equal that observed by Allrich et al. (1983) for boars of similar age. We observed a 1.3 to 1.7-fold increase in testosterone per mg tissue and a 1.55 to 1.59 increase in estradiol-17 $\beta$  per mg tissue after hCG compared with 2 and 3-fold increase, respectively, for both

steroids (fg/Lgdig cell) by Allrich et al. (1983) with a dosage of 625 to 3125 mIU hCG. Differences in steroidogenic capacity also were evident in vivo by the reduced concentrations of testosterone in the serum at 33 wk of age.

Our results suggest that feeding altrenogest to peripubertal boars delayed, but did not suppress permanently, reproductive development in the boar. Even though the testes of altrenogest-fed boars may have reduced ability to synthesize and secrete steroids after altrenogest withdrawal, sufficient secretion of LH and testosterone was available to support normal concentrations of testosterone and LH in serum and normal spermatogenic functions. These data suggest that the inhibiting effects of altrenogest were largely reversible in the boar.

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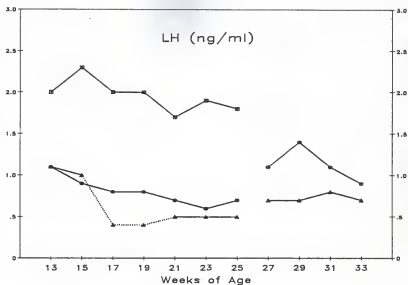


FIG. 1. Concentrations of luteinizing hormone (LH) in serum of barrows ( $n=18$ , -□-), control boars ( $n=17$ , -○-), and altrenogest (20 mg/d)-treated boars ( $n=18$ , -Δ-) from 13 to 25 wk of age. Broken line indicates a 6-wk period of altrenogest treatment. Standard errors are  $\pm 0.08$ . Concentrations of LH in serum of five control boars (-○-) and six boars previously fed altrenogest (-Δ-) also are shown from 27 to 33 wk of age. Standard errors are  $\pm 0.21$ .

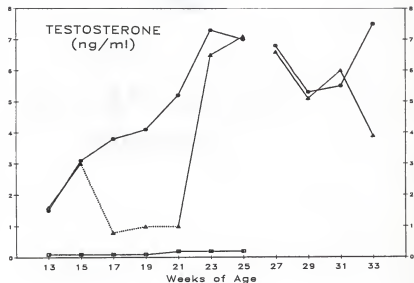


FIG. 2. Concentrations of testosterone in serum of barrows (n=18, -□-), control boars (n=17, -○-), and altrenogest (20 mg/d)-treated boars (n=18, -Δ-) from 13 to 25 wk of age. Broken line indicates a 6-wk period of altrenogest treatment. Standard errors are  $\pm 0.33$ . Concentrations of testosterone in serum of five control boars (-○-) and six boars previously fed altrenogest (-Δ-) also are shown from 27 to 33 wk of age. Standard errors are  $\pm 0.99$ .

TABLE 1. Gland weights at 25 wk of age after 6 wk of altrenogest feeding (20 mg/d) from 15 to 21 wk of age.

| Item                      | Control<br>boars   | Altrenogest<br>boars | Barrows          | SE   |
|---------------------------|--------------------|----------------------|------------------|------|
| Number                    | 12                 | 12                   | 12               |      |
| Paired seminal vesicle, g | 195.9 <sup>a</sup> | 65.1 <sup>b</sup>    | 3.6 <sup>c</sup> | 17.5 |
| Body of prostate, g       | 8.1 <sup>a</sup>   | 4.9 <sup>b</sup>     | 1.3 <sup>c</sup> | .7   |
| Paired bulbourethral, g   | 131.5 <sup>a</sup> | 92.7 <sup>b</sup>    | 4.3 <sup>c</sup> | 11.2 |

<sup>abc</sup>Means with different superscripts are different ( $P < 0.05$ ).



TABLE 2. Testicular and epididymal weights and sperm content at 25 and 33 wk of age after 6 wk of altrenogest feeding (20 mg/d) from 15 to 21 wk of age.<sup>a</sup>

| Item   | 25 wk              |                      |      | 33 wk            |                      |      |
|--|--------------------|----------------------|------|------------------|----------------------|------|
|  | Control<br>boars   | Altrenogest<br>boars | SE   | Control<br>boars | Altrenogest<br>boars | SE   |
| Number   | 12                 | 12                   |      | 5                | 6                    |      |
| Body wt, kg  | 113.6 <sup>b</sup> | 104.9                | 1.4  | 145.6            | 135.9                | 3.9  |
| Paired testes, g                                       | 590.7 <sup>b</sup> | 394.9                | 31.4 | 695.2            | 738.6                | 59.0 |
| % of total body wt                                     | .502 <sup>b</sup>  | .356                 | .003 | .48              | .55                  | .05  |
| Sperm·g testis <sup>-1</sup> , 10 <sup>6</sup>         | 25.1 <sup>b</sup>  | 17.7                 | 2.5  | 42.5             | 43.3                 | 6.6  |
| Total sperm·testis <sup>-1</sup> , 10 <sup>9</sup>     | 7.2 <sup>b</sup>   | 3.6                  | .8   | 15.4             | 15.4                 | 2.7  |
| Paired epididymides, g                                 | 110.5 <sup>b</sup> | 79.2                 | 4.9  | 144.1            | 146.4                | 12.5 |
| Sperm·g epididymis <sup>-1</sup> , 10 <sup>9</sup>     | 1.5 <sup>b</sup>   | .7                   | .2   | 1.3              | 1.4                  | .2   |
| Total sperm·epididymis <sup>-1</sup> , 10 <sup>9</sup> | 79.1 <sup>b</sup>  | 30.0                 | 8.6  | 96.8             | 98.6                 | 22.3 |

<sup>a</sup>Comparisons were made within age group (25 or 33 wk) only.

<sup>b</sup>Different from altrenogest-treated boars ( $P < 0.05$ ).

TABLE 3. Testicular histology of boars at 25 and 33 wk of age after 6 wk of altrenogest feeding (20 mg/d) from 15 to 21 wk of age.<sup>a</sup>

| Item                                   | 25 wk              |                      |     | 33 wk             |                      |      |
|--|--------------------|----------------------|-----|-------------------|----------------------|------|
|  | Control<br>boars   | Altrenogest<br>boars | SE  | Control<br>boars  | Altrenogest<br>boars | SE   |
| Percent                                |                    |                      |     |                   |                      |      |
| Epithelium                             | 51.0               | 50.4                 | .8  | 54.6              | 52.6                 | .2   |
| Lumen                                  | 20.5 <sup>b</sup>  | 24.6                 | 1.2 | 16.8 <sup>b</sup> | 23.3                 | 1.4  |
| Interstitial                           | 26.6 <sup>b</sup>  | 23.5                 | 1.0 | 25.0              | 27.5                 | 1.8  |
| Epithelial height x 10 <sup>-6</sup> m |                    |                      |     |                   |                      |      |
| Stage 1                                | 54.2               | 52.5                 | 1.9 | 60.9              | 57.1                 | 2.4  |
| Stage 8                                | 52.3               | 54.4                 | 1.5 | 65.6              | 59.5                 | 2.9  |
| Luminal diameter x 10 <sup>-6</sup> m  |                    |                      |     |                   |                      |      |
| Stage 1                                | 70.0               | 75.6                 | 2.9 | 73.4              | 71.9                 | 3.4  |
| Stage 8                                | 73.0               | 77.4                 | 3.6 | 74.7              | 77.3                 | 7.6  |
| Tubular diameter x 10 <sup>-6</sup> m  |                    |                      |     |                   |                      |      |
| Stage 1                                | 178.5              | 180.4                | 4.2 | 194.6             | 186.2                | 4.6  |
| Stage 8                                | 177.5              | 185.7                | 5.6 | 205.9             | 196.8                | 5.7  |
| Epithelium g/testis                    | 154.9 <sup>b</sup> | 100.9                | 7.9 | 192.9             | 197.9                | 15.4 |
| Interstitial g/testis                  | 81.6 <sup>b</sup>  | 46.0                 | 5.6 | 93.9              | 93.6                 | 10.8 |

<sup>a</sup>Comparisons were made within age group (25 or 33 wk) only.<sup>b</sup>Different from altrenogest-treated boars (P<0.05).

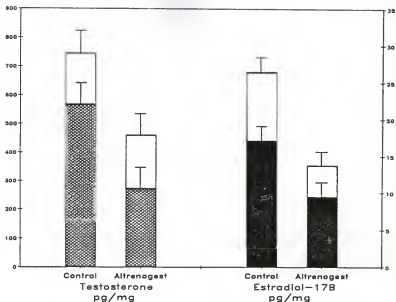


FIG. 3. In vitro production of testosterone and estradiol-17 $\beta$  by testicular pieces incubated in TC199 in the presence (5,000 IU) or absence of human chorionic gonadotropin (hCG). The stippled or colored portion of the bars illustrates steroid production without hCG. Additional steroid production in the presence of hCG is shown by the clear portion of each bar.

ALTRONEGEST INFLUENCES GROWTH, REPRODUCTIVE, AND CARCASS TRAITS IN  
MALE SWINE

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AN ABSTRACT OF A MASTER'S THESIS

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Three experiments (Exp.) were conducted to determine the effects of a synthetic progestogen, altrenogest (AL), on body growth, carcass and muscle quality, testicular function, and reproductive development. In each experiment, litter was used as the block and male pigs were assigned randomly to treatment groups. Male pigs were fed a medicated, 18% protein (.95% lysine) milo-soybean diet fortified with vitamins and minerals. Treated boars received a daily dose of 20 mg AL.

In Exp. 1, AL was fed (beginning at 15 wk of age) to 24 crossbred boars from six litters to determine effects on body growth and testicular characteristics. Treatments included: 1) control boars (CB), 2) AL fed for 3 wk, 3) AL fed for 6 wk, and 4) AL fed for 9 wk. Average daily feed intake, average daily gain, final backfat thickness, and weight at 24 wk of age were similar among treatment groups. Weights and volumes of paired testes at 24 wk of age decreased linearly ( $P < .001$ ) with increased duration of AL feeding. Concentrations of testosterone and estradiol in testicular venous serum collected at castration were lower ( $P < .01$ ) in AL-treated boars than CB. Altrenogest feeding for 3 and 6 wk maintained lower ( $P < .05$ ) peripheral concentrations of testosterone in serum than in CB from 15 to 24 wk of age. Concentrations of testosterone in serum remained below 1 ng/ml during AL treatment and appeared to increase after AL withdrawal in the 3- and 6-wk treatment groups, but did not reach control concentrations. Testosterone in serum of the 9-wk group never exceeded 1 ng/ml. These data suggested that feeding AL inhibited both growth and normal steroidogenesis of the testes without altering body growth.

In Exp. 2, AL was fed to crossbred boars to determine effects on growth, carcass traits, and "boar odor." Male pigs ( $n=54$ ) from 18 litters were assigned at 15 wk of age to three treatments: 1) control boars (CB), 2) boars fed AL for 6 wk followed by 4 wk with no AL (AB), and 3) barrows (BA) castrated at 2 wk of age. Control boars were more ( $P < .05$ ) feed efficient than BA, but average daily gain was

similar in both groups from 15 to 25 wk of age. Boars fed AL gained more ( $P<.01$ ) weight than BA during treatment. Feed intake and average daily gain were less ( $P<.01$ ) for AB than CB and BA during the 4 wk withdrawal period. Casual daily observations of AB indicated intense mounting activity beginning 5 d after withdrawal of AL. Boars previously fed AL weighed less ( $P<.01$ ) than CB and BA at 25 wk of age. Hot carcass weight of AB was less ( $P<.05$ ) than CB and BA. Control boars and AB had greater ( $P<.05$ ) percent muscle and less backfat than BA, whereas CB had the largest ( $P<.05$ ) loin-eye area. Intensity of "boar odor" in fat samples was similar in CB and AB and was greater ( $P<.01$ ) than that of BA based on sensory panel evaluations. Weights of the submaxillary glands was similar for AB and BA, but both were lighter ( $P<.05$ ) than those of CB. These data suggested that feeding AL to growing boars had no negative effects on carcass quality traits nor on growth performances during treatment. It is possible that "boar odor" was prevented during AL treatment, but it evolved in association with increased testosterone in serum of boars after AL withdrawal.

In Exp. 3, crossbred boars were used to evaluate the influence of AL on reproductive development. Treatments included: 1) control boars (CB,  $n=18$ ), 2) boars (AB,  $n=18$ ) fed AL from 15 to 21 wk of age, and 3) barrows (BA,  $n=18$ ). Twelve pigs from each treatment were slaughtered at 25 wk of age (4 wk after AL withdrawal) and the remainder of the CB ( $n=5$ ) and AB ( $n=6$ ) were castrated at 33 wk of age (12 wk after AL withdrawal). Paired seminal vesicles, body of prostates, and paired bulbourethral glands weighed less ( $P<.05$ ) in AB than CB, but those tissues were heavier ( $P<.05$ ) in AB than BA. Testicular and epididymal weights and content of sperm were less ( $P<.05$ ) in AB than CB at 25 wk of age, but were similar at 33 wk of age. Total seminiferous epithelial tissue and total interstitial tissue per testis were less ( $P<.05$ ) in AB than CB 4 wk after withdrawal of AL. Twelve weeks after withdrawal of AL, total seminiferous epithelial tissue and total interstitial tissue per

testis were similar for CB and AB. Total and luminal diameter and epithelial height of seminiferous tubules at Stages 1 and 8 were similar for CB and AB at both ages. Concentrations of testosterone and LH in serum were similar for CB and AB prior to AL treatment. Testosterone and LH were suppressed in AB during treatment, but rose to levels similar to CB at 2 wk after withdrawal of AL and were similar for all boars from 23 to 33 wk of age. These data suggested that feeding AL to prepubertal boars delayed, but did not reduce permanently reproductive development.